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PNEUMOTHORAX; A HISTORICAL, CLINICAL, AND EXPERIMENTAL STUDY.

By CHARLES P. EMERSON, A.B., M.D.,

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Definition.

By "*pneumothorax*" we mean the presence of gas in the pleural cavity. This condition has been termed also *pneumato-thorax* (Piorry [42]) or *pneuma-thorax* (Johnson [56]). This was first written as a compound word, but more lately has the hyphen been omitted.

The term is only in rare cases strictly appropriate, since usually there is also present a liquid exudate, hence the names *hydro-pneumothorax, pyo-pneumothorax, hæmo-pneumothorax;* or to signify that the liquid was not the primary element in the condition, *pneumo-hydrothorax,* etc. Since it is the presence of gas which occupies our attention in this paper we shall use the simple term "pneumothorax."

CHAPTER I.

LITERATURE AND HISTORY.

Introduction.

The following method of presenting the literature and history of a subject is rather an experiment. We confess to receive little or no aid from the chapters usually prefacing articles, except that there we find a few references to other articles. The long lists of names of those who agree or disagree on a certain point give us little idea of what these several men found or did not find, and still less of the relative value of their contributions. Although an abstract is never a substitute for the original article, yet it is more interesting reading than simply the author's name and the title; and the following, it is hoped, will give the reader some idea as to the articles which are worth his careful perusal in the original.

The paragraphs in fine print are either direct quotations, or condensations, or very free translations of the article in question. Sen-

tences in parentheses are our comments on the same. Paragraphs in coarser type are remarks on the article as a whole.

Older Writers.

The first sign of pneumothorax which we find on record is the splashing sound produced by succussion, mentioned in The Complete Works of Hippocrates. This sign is often referred to as "Hippocratic Succussion," yet it is well to remember that the succussion is not the sound but the shaking which produces it. This sign is referred to in the above mentioned works several times. It was supposed to be a sure sign of empyema. Yet none of these references are in those books which critics are willing to admit belong to the genuine works of Hippocrates.

1. Hippocrates. *5th Century, B. C. Littré's translation—Œuvres complètes d'Hippocrate,* 1844-1851.

Vol. VII, "The Diseases," p. 71. After giving the picture of a very ill person the author says, "Seat him on a seat which will not stir. Let some one hold him by the arms while you shake him by the shoulders, and listen to hear on which side the sound is produced." He advises to operate on that side, but states there is less danger to incise the left.

P. 67. Succussion is here recommended not for diagnostic purposes but as a therapeutic measure, in fact as one of the gentler, less heroic means to be first tried, to encourage the rupture of a peripneumonic abscess. (One cannot wonder that later writers condemned all succussion as too severe.)

P. 153. The description here is of an abscess of the lung breaking into the pleura. The patient seems at first to recover since the pus occupies a larger cavity, but this soon becomes filled and the symptoms are then worse. The advice given is to aid expectoration in hopes of thus evacuating the pus. This failing, by succussion determine which is the affected side, and open this by incision or cautery.

Vol. VI, p. 165. "One does succussion, and the wave of pus strikes the wall of the chest: this patient cauterize or incise . . ."

P. 171. In effusion of the abdomen "the diagnosis is more difficult since one can not assure himself of it by succussion."

(The above five quotations are from the books on "The Diseases" which "Almost all modern authors . . . concur in rejecting as spurious" [Adams]. Hence Hippocrates is denied their authorship.)

Vol. VI, p. 307. "The Places in Man." The author is speaking of empyema following ruptures and contusions of the flesh, and says, "If one tries succussion there is produced a wave and a sound; it is there that one should cauterize."

P. 309. "If the eruption of pus has not yet occurred, succussion produces a noise in the side as in a leathern bottle."

(But it is "uncertain whether Hippocrates wrote this book or not.")

Vol. V, p. 681. "Coacæ prænotiones." "Those empyema cases who when shaken by the shoulders produce much sound have less pus in them than those who produce little sound. The latter also have more dyspnœa and their face is more discolored. Finally, those who produce absolutely no sound; they have great dyspnœa, and livid finger nails; they are full of pus, and their case is desperate."

(Laennec remarks that the writer of this sentence almost discovered pneumothorax and would have, had he had enough cases, and noticed why those with least pus made the most noise on succussion. Yet this book also is denied Hippocrates. Adams says, "The Coacæ prænotiones have very little ancient authority in their favor." "Of late years the opinion has gained pretty general assent that these two treatises are more ancient than the days of Hippocrates, that in fact they constitute the materials out of which he composed the 'Prognostics' and are the results of the older observations made by the priest-physicians in the Asclepion, or Temple of Health at Cos." We claim no right to discuss this question of age but nevertheless would ask these critics if these similar descriptions of succussion would not be evidence that the books in which they are found were either all before or all later than Hippocrates, granting that he made no mention of it.)

In conclusion, the writers of the above mentioned books formerly ascribed to Hippocrates, certainly describe cases of pneumothorax under the name empyema, for the splash on succussion occurs in no other disease. They did not consider it an infallible sign, for when it fails they advise to incise the distended, or the painful side. They were correct in considering it a sure indicator of the affected side.

Another point of importance is that they were dealing chiefly with cases of pneumothorax following disease of the lungs, for until the beginning of the last century such cases were for the most part overlooked, and traumatic cases alone were described and misinterpreted.

There has been considerable discussion as to what disease it was that caused a pleural effusion which on succussion would splash. If, as the descriptions would indicate abscess of the lung is meant, then Littré may be right in saying it was perhaps more common in that age and climate than now. Adams, however, thinks that under the term abscess were included the tuberculous areas, especially of acute phthisis, which in the age and climate of more recent writers is certainly common enough.

But pneumothorax is a rare condition when compared with empy-

ema, and the number of cases of this latter disease probably shaken hard by the shoulders without eliciting a sound evidently brought this sign into disrepute, for Laennec states that he carefully read the commentators of Hippocrates and found no indication that it was used by one of them. Some record that they heard it, but as a diagnostic sign its value was denied. They operated quite fearlessly in empyema, some advising incision, some the red hot iron, which latter method must have furnished plenty of cases of post-operative pneumothorax, but the procedure underwent little change till Celsus (1st century) (*Sprengel, Histoire de la méd.* Vol. 9, De l'Operation de l'Empyème, 1820), who described more precisely the method to be used in empyema following fracture of the ribs.

2. Celsus. De re medica, etc. Translation by Jas. Grieve, M. D., 1837. Chapter IX. "But if a rib be entirely broke through the case is more troublesome; for both vicient inflammations, and a fever, and suppuration, and often a danger of death ensue, and a spitting of blood." "If (after gentler means) the suppuration take place . . . no time must be lost . . . but in the part where it shall swell most a hot iron must be introduced till it reach the pus, which must be evacuated."

Hence we find nothing in Celsus which indicates pneumothorax, yet this is of interest since the symptoms of pneumothorax are similar to those of empyema, but empyema seldom follows fracture of the ribs, hence some of Celsus' cases may have been pneumothorax. From this time on the cases of pneumothorax described followed fractured ribs or other trauma of the chest wall, but it was the emphysema resulting that attracted attention; the air in the pleura was for the most part overlooked, if indeed there was air in the pleura, for a lung may be pierced by a broken rib, and emphysema of the chest wall follow without the development of pneumothorax. But in some cases pneumothorax seems to have been present, judging by the clinical course, hence we shall consider the emphysema as indicating the possibility of air in the pleura.

"The first reference to emphysema as a complication of fractured rib" (Demarquay) is found in

3. Paré. Les Œuvres d'Ambroise Paré, Chap. XII of Edition 8th, 1628, where as a complication of broken ribs he says, "The flesh is swollen as if inflated and when pressure is made on it with the hand the air can be felt to disperse."

A very early discussion of the effect of air in the pleural cavity is by

4. Purmann, *Chir. Lorbeerkranz,* 1692, (quoted from Sprengel), who maintained that all criticisms of the operation for empyema are groundless, since military surgeons see a great many penetrating wounds of the chest which are not fatal, and in which the penetration of atmospheric air does not cause inconvenience.

One of the first cases in which air was noted to come from the pleural cavity is given by

5. Riolan, ENCHEIRID-ANAT. lib. iii, cap. ii., 1648 (quoted from Laennec), who stated that he had several times seen paracentesis successfully performed on patients considered as affected by dropsy, but from whose chest only air escaped. (Hewson believes that one case cited by Riolan is an example of air in the pleural cavity resulting from disease of the lung rather than from external injury.)

6. Bonet, t. I, p. 506, Obs. 54, 1670 (quoted from Devilliers), reported a case of emphysema of the lungs and, giving Riolan's case as an example, says in such a case one should perform paracentesis.

During this same year is reported one of those cases which were a stumbling block to many in the way of a clear understanding of the part played by the elasticity of the lung.

7. Fabrice de Hildan Cent. II, Obs. 32, p. 108, 1670 (quoted from Boyer, Vol. VII, p. 264), reported a case of hernia of the lung. A man received a sword wound between the fifth and sixth ribs near the sternum through which a part of the lung projected. This portion was excised with a red hot iron and the lung pushed back into the chest. To do this, however, he must enlarge the wound. Recovery was rapid and complete.

A similar case is reported by

8. Ruysch, *Obs. Anat. Chirurg.,* 1691, Obs. 53, p. 50 (quoted from Boyer). A man was wounded in the anterior-inferior part of the chest and hernia of the lung resulted. A surgeon, thinking it the omentum which was extruded, applied a stout ligature. Ruysch, who was consulted, saw the error, but advised not to molest the wound further, that the ligated tissue might separate. This followed and the wound healed.

A much quoted case of pneumothorax, interesting in many particulars is that of

9. Littré, *Mém. de l'Acad. des Sc.,* p. 10, 1713 (quoted from Proust, Demarquay, and Boyer).

A man, thirty years old, received a sword thrust in the chest, and refusing operation for empyema died in five days. After the injury there

developed subcutaneous emphysema, and he became of monstrous size. The emphysematous skin was eleven inches thick over the front of the chest, nine inches thick over the abdomen, six on the neck, and four over the rest of the body. The emphysema involved the entire surface of his body except the palms of the hands, soles of the feet and the scalp.

He was able to breathe only by making the most violent efforts. He expectorated blood and was therefore bled six or seven times.

At the autopsy an incision was made into the chest from which escaped considerable very stinking gas. The chest was then opened. About two pints of "decomposed blood" was found. The wound of the third lobe of the right lung was still open. This lobe was firm and blackish, the other two soft and a little inflamed.

The interest in this case lies in the certainty that it was pneumothorax and the great extent and degree of the emphysema. Laennec[41] considers it a case of pneumothorax arising from decomposition of blood, hence a case of essential pneumothorax (i. e. the air arose in the chest.) Proust,[124] however, calls attention to the fact that the external and internal wounds existed, hence it must be considered a case of traumatic pneumothorax.

That same year a similar case was reported by

10. Méry, *Ann. de l'Acad. des Scienc.*, 1713, p. 116 (quoted from Boyer). A man, 60 yrs. old, was run over, the accident breaking the 4th and 5th ribs of the left side. The dyspnœa and emphysema increased till his death on the fourth day. At the autopsy the emphysema covered all the body but the palms of the hands and the soles of the feet. On opening the chest no blood was found. The pleura was a little torn. (No mention was made of the presence of air.)

A case almost unique was reported by

11. Boerhaave, in 1724 (quoted from Saussier). PNEUMOTHORAX FOLLOWING RUPTURE OF THE ŒSOPHAGUS. The famous Dutch Admiral Wassenaer, striving to vomit after a heavy meal, as was his custom, experienced a sudden atrocious pain and sensation of something rupturing in the upper part of the abdomen. He died in great agony. The cavity of the chest contained a great amount of air which escaped with force. The lungs were compressed, discolored, and swam in a fluid like that found in the stomach. In the left cavity three fingers' breadths above the diaphragm was a tumor mass three inches in diameter formed of loose cellular tissue . . . in the interior of which were the two ends of the œsophagus completely torn across.

12. Le Dran, *Obs. de Chirurg.*, 1731, t. I, Obs. 29, p. 195. (Quoted from Boyer.) EMPHYSEMA FOLLOWING FRACTURED RIB. A man was kicked by

a horse in left breast. The next day the emphysema was even four fingers' breadth deep. He suffered from great dyspnœa and cough. Recovery followed.

One of the most interesting items of this period concerning wounds of the chest is given by

13. G. Marequest de la Motte, 1732 (quoted from Sprengel). He wrote that it was the custom at that time to suck all wounds, especially those of the chest. At every duel a professional sucker was present. So successful was this treatment that some of the cures were attributed to the devil, and the priests refused extreme unction to persons thus treated. He thought, however, that suction would be very dangerous when in the interior of the body there might be an open vessel, hence he rejected it for penetrating wounds of the chest, that is in precisely those conditions for which all others praised it the most.

It is hard to see why such treatment should be especially popular for penetrating wounds of the chest, unless the value of aspirating the pleural cavity was unconsciously recognized.

In the next decade was published the work of

14. Haller. De respiratione esperimenta anatomica, 1746, which Devilliers states victoriously combated the general belief in the physiological normal presence of air in the chest.

15. Cruveilhier. Anat. path., 1747, t. I, p. 161 (quoted from Saussier). Case of extra-pleural emphysema. The patient in a spasm of coughing felt something rupture in the chest; dyspnœa resulted, and emphysema of the neck, axillæ, and chest. At autopsy the pulmonary pleura showed the changes now termed interstitial emphysema, which the author correctly attributed to ruptured pulmonary vesicles and the subsequent entry of air into the interlobular tissue.

This case is here cited not because of the possibility of pneumothorax, but as an early case showing the protecting action of the visceral pleura in cases where the lung is ruptured.

16. Combalusier. Pneumato-pathologia, 1747, p. 508. (Although this case is given on hearsay evidence, it is nevertheless of interest.) Combalusier said that Sidobre told him that his uncle M. Berbeyrac, the celebrated physician, "one day made a puncture into a patient's chest whom he thought suffered from purulent empyema; but there escaped not pus but air which rushed out with a noise; and in consequence the person got perfectly well."

A case of pneumothorax historically quite important is that of

17. Meckel. Sur de l'Air répandu ramassé dans la cavité du thorax qui arrêtait la Respiration et qui a causé la mort. *Mém. de l'Acad. roy. de Sc. de Berlin*, 1759, XV. (Quoted from Proust). "A soldier 18 years of age was in the hospital 10 days complaining of sensation of extreme anxiety and difficulty of respiration. He could breathe only when sitting upright in bed." Despite all treatment he died. Before entering hospital he had spent seventeen weeks in bed because of difficulty of respiration for which he could assign no cause. "I performed the autopsy. Before opening the thorax I found the liver depressed . . . obliquely, being higher on the left side and lower on the right. The diaphragm extended convex downward into the abdominal cavity on the right side . . . in such a manner that it appeared inflated like a bladder below the cartilages of the seventh and eighth ribs. For this reason the liver . . . was situated in the right iliac fossa. . . . I expected . . . to find the cause of this phenomenon pus or water exuded into the cavity of the thorax, especially considering that the diaphragm bulged into the abdomen with the appearance of a bladder full of water. But what was my surprise on opening the diaphragm when air issued from the chest . . . and the diaphragm collapsed. In the cavity of the thorax on the right even as high as the third rib was an empty space which the lung did not fill but which was free, dry and lined throughout by the pleura, which was whitish and only a little thicker than normal . . . The lung was dry, dense, firm; it contained no air . . . (there were) a few adhesions on the anterior surface to the third rib . . .it could not be inflated from the trachea (which he ascribed to a plug of mucus in the bronchus). Hence the air enclosed in the right lung was rarified by the warmth (of the body) burst its walls . . . into the cavity of the thorax and there expanding . . . compressed the lung, and prevented the circulation of the blood as well as of the air, which caused the feeling of anxiety, the difficulty of respiration, the frequent pulse and finally the suffocation with the death.

"This very rare observation is not useless to refute the opinion of the presence of an elastic fluid between the lung and the pleura, and necessary for the mechanism of respiration. It is, rather, clear . . . that the expanded air between the pleura and the lung, far from aiding respiration, placed there an obstacle by compressing the lung."

This case is important since the author recognizes the presence of the air in the pleura, and its effect on the respiration. This case was early (Devilliers q. v.), quoted as one of essential pneumothorax until Proust's paper.

In connection with the following abstract we shall consider again the question of succussion as a sign in diseases of the chest. This had fallen into disuse since the earliest Greek writers.

18. Morgagni. De Sed. et Caus. Morb., 1760, Ep. XVI, Art. XXXVI (transl. by Alexander, 1769). A woman in the fiftieth year of her age had

been for many days troubled with a difficulty of breathing attended with wheezing; no orthopnœa; painful œdema of one arm of the kind sometimes observed in dropsy of the thorax." The patient said that "upon motion of the thorax she felt a fluctuation in it, and a weight even without motion." She died suddenly. Fluid was found in each pleural cavity.

Speaking of signs of fluid in the chest, Morgagni says, "And indeed we could easily do without all of them if that one sensation of a fluid fluctuating in the thorax were perpetual, without any cause of suppuration having preceded, for sometimes, indeed, that this fluctuation is not only perceived by the patients themselves but even heard by others Hippocrates has evidently asserted (quoting passages on succussion) . . . And that is confirmed by more than one of the observations of modern physicians such as I have mentioned above, such as the celebrated Fantonus published of his father's; this of mine depending upon the feeling of the patient only; and also those of the celebrated men, John David Manchartus and John Philip Wolffus. But this symptom neither is nor can be perpetual; for besides that you will look for it in mine, and Valsalva's observations, in vain, the same also happens in most of the observations of other persons that you will read, who, it is to be supposed, could not have passed over this clear and evident mark of the disease had it really existed, and you will even find it denied by some that it ever existed. So that in that virgin and in that Polish senator whom I mentioned above, to omit others at present, notwithstanding there was such a quantity of water in the thorax, you will read in their histories that there was no fluctuation while they were living." He argues that there can be no splash when there is very little or very much fluid present, and there should be a time when there is just enough, "but some do not notice it, . . . some do not attend to it, others finally do not discover it to the physicians; and the symptoms may at the same time be so few, so slight, and so common to other diseases, that if one inquires into this symptom he may be thought to do it without reason; but to lay hold of patients by the shoulders and shake them, or agitate them in any other way, is certainly not practicable in all patients."

Morgagni also cited the case of a child who died from rupture of the lungs without fracture of the thorax. In Exp. XVI Morgagni mentions a case (Stokes) which presented a peculiar onset, since there was the sensation of something giving way, and a dropping into the chest perceptible not only to the patient but also audible by the bystanders.

Morgagni's opinion is therefore that in cases of effusion into the pleural cavity the splash on succussion is too rare to be of value, also that the disturbance to the patient does not justify its trial.

Laennec adds the names of Willis and Paré to those of the above list who stated that they had heard the succussion splash.

In the chapter De l'Operation de l'Empyème, Sprengel says:

10. "**Henri Bass** was the first to advise that the skin be drawn as much as possible up before opening the chest for empyema that the wound of the external pleura might not coincide with that through the skin," and "Various illustrations would have proven that the chest could be opened with benefit not only when liquid exudate was in that cavity but also in *internal emphysema.*"

20. **Heuermann** had at least pointed out this latter fact, but it was

21. **Alexander Monro the younger**, who the first in 1760 proposed the operation as a therapeutic measure for this trouble. (He proposed it in his lectures but evidently did not publish it.)

22. **Benjamin Gooch** adopted his opinion after seeing, following a paracentesis of the chest performed for a suspected empyema a great deal of air escape and the pain disappear with the oppression."

An observation which for English writers was of importance is that of

23. **Hunter (William).** The History of an Emphysema. *Medical Observ. and Inquiries*, Vol. II, 1762.

(This communication seems to have led Leake, Huxham and Russell to report their cases, and Hewson to study the problem of emphysema and its importance experimentally.)

It was the case of a man injured in the side by a fall from a horse. "I visited him . . his form was that of a human skin stuffed. I have often seen such anatomical preparations and wondered at so much pains being taken to so little purpose; for till I saw this poor man they seemed hardly to resemble anything human." On striking him with the hand "it sounded like a wet drum." He suffered from difficulty in breathing which increased in proportion as the skin became more elevated and tense. It was laborious as well as frequent. Inspiration was short and almost instantaneous and ended with the catch in the throat produced by shutting the glottis; after this he strained to expire for a moment without any noise, then suddenly opening the glottis he forced out his breath with a sort of groan and in a hurry and then quickly inspired again; so that his endeavor seemed to be to keep his lungs always full. He said his difficulty in breathing was owing to an oppression or tightness across his breast near the pit of the stomach. The sputum was blood tinged. Incisions were made and the air reduced by "stroking." "The emphysema was quite gone in a few days and in about two months he was in all respects well."

But Hunter does not consider the air in the pleura (if indeed there was any). He feared emphysema of the lungs and was surprised to see the symptoms disappear as the external emphysema reduced.

Discussing the formation of such emphysema he says, "Now I apprehend that it can be only so (formed) during expiration, for in the time of inspiration the air will rather be drawn inward at the wound of the

pleura than pressed outwards; the expansion of the cavity making a suction on all hands, as we see in penetrating wounds of the chest. Monsieur Méry indeed is of the opinion that the air is forced into the cellular membrane during expiration only."

The next paper is of such importance that the abstract will be longer than may seem necessary, but it is a paper which seems to have been much neglected or misquoted by later writers. To us it seems one of the most important.

24. Hewson. The Operation of the Paracentesis Thoracis proposed for Air in the Chest; with some Remarks on the Emphysema, and on Wounds of the Lungs in general; By William Hewson, Reader of Anatomy. Communicated by Dr. Hunter. Read June 15, 1767. *Med. Observ. and Inquiries*, Vol. III.

For emphysema following wounds of the chest Hewson proposes the operation of " paracentesis thoracis in order to let the air out of the chest, for that the confinement of air in that cavity occasions the worst symptoms in that disorder, and even death itself I am almost persuaded." " This inflation of the cellular membrane has been commonly looked upon as the most dangerous symptom of the disease. . . . It is natural to suppose that the wound of the pleura and intercostals may sometimes be too small to suffer the air to get readily into the cellular membrane and to inflate it, but may confine a part of it in the cavity of the thorax so as to compress the lungs, prevent their expansion, and cause the same symptoms of tightness of the chest, quick breathing, and sense of suffocation, which water does in the hydrops pectoris or matter in the empyema." As clinical proof of this he cites the cases of Littré, Méry, Hunter, and Cheston. Méry's, Littré's and Hunter's cases have already been cited.

25. Cheston's was that of a man who as a result of a blow on the chest received the fracture of the 10th and 11th ribs, which wounded the lungs. He had a constant sense of suffocation, cough and blood-tinged sputum. He died on the fourth day. At autopsy the lung was found wounded opposite the broken ribs.

" Now were not," Hewson says, " the symptoms in these cases such as might be expected to arise from air contained in the cavity of the thorax? I am inclined to believe they were; notwithstanding that authors in general account for them otherwise . . . I was thence induced to believe that this operation (paracentesis thoracis) might be performed for letting out the air with some probability of success. But as in such cases no air had hitherto been actually discovered in the cavity of the thorax, it therefore might be doubted whether the symptoms were not owing to some other cause . . ." Hence he conducted the following experiments:

Exp. I.—Rabbit.—The skin was pushed well to one side, and a sharp knife thrust into cavity of thorax, and moved about to wound lung. The skin was then allowed to slip back into place, that the skin wound might not be opposite that of the chest wall, and a bandage applied. He expected

emphysema would develop but, although he tried three or four times, this did not occur. He killed the animal and found the wounds closed by hæmorrhage into the lung tissue.

Exp. II was similar to I but on a dog, and with the same result. He therefore concluded that to let air out of the lung into the pleural cavity a puncture or incision was improper, and that in emphysema cases the injury to the lung must have been a superficial abrasion or laceration.

Exp. III.—Similar to above, but using a blunt probe to lacerate the lung. No emphysema resulted.

Exp. IV.—He opened the pleural cavity of a rabbit in such a way as not to injure the lung, blew air into cavity, and confined it there by a bandage. The animal breathed more frequently and laboriously. On removing compress the air rushed out and the animal recovered.

Exp. V.—Tried the same on a dog, but the struggling of this animal rendered the experiment a failure.

"I made no further experiments, being indeed almost convinced from these that it was the air confined in the cavity of the thorax which had occasioned the violent symptoms in the cases mentioned above, and not the mere wound of the lungs." Very soon he saw a case which "fully satisfied him." A young man to escape death threw himself from the second-floor window of a burning house and fractured his skull. In the evening he was emphysematous, breathed with difficulty, and died. On opening the abdomen the diaphragm was depressed and loose on the right side. On puncturing the thorax air rushed out. The lung was found much collapsed. No blood or lymph was in the cavity, hence much air must have been. There was a fracture of the first rib, but no wound of the lung opposite it. The wound of the lung was found on the under surface of the lower lobe at the middle of the surface which lies against the diaphragm. There the pleura was raised "into vesications about the size of one's nail" filled with air. Here was the opening from which on inflation the air escaped. The wound was therefore at some distance from the fractured rib.

Since paracentesis thoracis is so successful when fluid or pus is in the chest where the lungs are much diseased, why should it not be more successful when the effusion is air and where the lungs are as a rule normal? In wounds of the lungs when symptoms of tightness and suffocation come on "so far should we be from dreading the emphysematous swelling of the cellular membrane that we should rather consider it as a favorable symptom, showing that the air is not likely to be confined in the thorax; and so far should we be from compressing the wound to prevent the inflation or emphysema that we should rather dilate it or perform paracentesis."

"It has been observed that in gunshot wounds where the ball has passed through the lungs, and in other large penetrating wounds of the chest, the patient has breathed more easily when the external wound has been covered and has hardly been able to breath when it was open. In

these circumstances the difficulty of breathing seems to be owing to the air getting into the cavity of the thorax in inspiration instead of entering the lungs by the trachea so that the lungs are not distended by the expansion of the chest; and the patient, whilst such a wound is uncovered, is deprived of the use of the lobes of that side either partially or intirely according as the wound of the thorax bears a greater or less proportion to the branch of the trachea of that side in which the wound is."

The case he reports shows also "that the lungs may be lacerated and the air let loose without any wound penetrating the cavity of the thorax; for in that instance the laceration was at a distance from the broken ribs and was produced by the violence of concussion only. It therefore intimates the necessity of the operation even in some cases where there is no emphysema . . ."

"As vomicæ and ulceration of the surface of the lungs are attended with erosion and destruction of the air vesicles it may at first sight seem probable that they may also be attended with a discharge of air into the cavity of the thorax . . . Whether air has ever thus been let loose into the cavity of the thorax so as to suffocate the patient, I cannot take it upon me to determine, but abscesses of the lungs with adhesions to the parietes of the thorax have been known to be attended with the emphysema." (He cites Palfyn's case, *Anat. Chirurg.*, p. II, c. 20; Riolan's, which he thinks resulted from disease of the lung; and Hunter's.)

We will leave the dispute between teacher and pupil for those interested to solve (see Monro[25]). Hewson's contribution is not small. The presence of air in the pleura he recognized, and its importance appreciated (as did Meckel[17]), but he gives meager but still good experimental evidence for the same. The case which he reports is one of a very few reported of rupture of the lung. He understood the harmless nature of emphysema. He also recognized the importance of the ratio in size between the external wound and the bronchus in the result on respiration.

John Bell[35] says of this work, "Hewson, again, is as vain of this trifle as if he were not rich in discoveries really honourable." Yet this trifle was interesting enough for him to spend some space on its explanation.

In the same volume as the above, and evidently inspired by Hunter's communication, is one from

26. Leake. "The History of a Fractured Rib, with an Emphysema in most Parts of the Body. By William Leake, Surgeon to the Garrison of Plymouth, with a Letter of Dr. Huxham, F. R. S., to Mr. Leake, on Emphysematous Cases from internal causes, communicated by Dr. Hunter." *Med. Observations and Inquiries*, 1767, Vol. III.

A soldier, 60 yrs. of age, fell on the spiked points of the palisades. No external wound found. There were fracture of a rib on left side, and an

emphysematous tumor the size of a crown piece. That evening the progress of the emphysema was astonishing, involving about half the trunk. Following Hunter's advice, "a longitudinal incision was made of about an inch and a half, above the fracture, where the swelling appeared most prominent. The air immediately rushed out with noise and violence." The emphysema was reduced by "stroking and pressing it." The next morning, as he was uneasy and the swelling greater, the bandage was again removed and the emphysema reduced by stroking it towards the orifice. Patient was up and about in five or six days. The writer considers that the symptoms were due to the subcutaneous emphysema.

Dr. Huxham's Letter. (He wishes to "go Leake a little better," for he says) "Indeed an emphysema doth not uncommonly happen on a fracture of the ribs and a laceration of a lobe of the lungs. But an emphysema of a large extent, without any kind of laceration of the lungs or fracture of a rib is not so common nor so easily accounted for. The following case is, I think, somewhat singular." A sailor was seized "with putrid fever and sore throat." On the seventh or eighth day of the disease an emphysematous swelling appeared in his face, neck, and all over his breast, especially on the right side. Careful examination showed it to be "a compleat emphysema." This totally vanished in a few days and he soon recovered from the fever. "I am persuaded that this more frequently happens in putrid malignant fevers than is commonly imagined, and it is not improbable that elastic air may be generated even in the arterial and venous system and be productive of terrible symptoms, vast oppression, anxiety, palpitation, intermitting pulse, delirium, etc. . . ."

We can forgive Huxham his explanation of such emphysema as "generated merely by the putrescence of humours" but would like to know what the trouble with his patient was. Rather than doubt the evidence of his fingers' tips we can only imagine a rupture of the lung without that of the pleura, the gas dissecting its way into the neck as is the rule in such cases.

In this same volume is another similar communication from

27. Alexander Russell. *Med. Obs. and Inq.*, 1767, Vol. III. A letter to Dr. Wm. Hunter describing a case of extreme emphysema of the entire body excepting the legs, following fracture of the sternum, clavicle and arm. A small incision was made over the most prominent part of the thorax. A large amount of air escaped; breathing was much easier. The patient recovered.

28. Alexander Monro. A State of Facts concerning the first proposal of Performing the Paracentesis of the Thorax on account of Air Effused from the Lungs into the Cavities of the Pleuræ. In Answer to Mr. Hewson. Edinburgh, 1770.

He states that Mr. Hewson attended his course of lectures 1761-2, during

which he advised the above operation for air in the pleura. (The most of this paper is an arraignment of Hewson for his claim to have discovered lacteals, a subject on which he heard Monro lecture.)

(Benjamin Bell states that Monro advised this procedure in 1760 in his lectures before the University of Edinburgh.

Hewson later admitted he probably did hear him lecture on this point.)

That all emphysema of the chest wall is not caused by air from the lungs is claimed by " the celebrated "

29. Jean Louis Petit. 1774. (Quoted from Warme.) This surgeon claimed that even if the wound did not reach the cavity of the chest it could still cause emphysema, for a sudden inspiratory movement, that is, the sudden elevation of the ribs, can suck air into the wound, and this air finding no opening in the chest to enter will be forced to spread into the tissue of the chest wall.

30. Kellie in 1775 (quoted from Sprengel), in a case of considerable emphysema accompanied by violent oppression, performed paracentesis in the manner recommended by Monro. There escaped with a rush a great amount of gas. The oppression ceased; the patient recovered.

31. Pouteau. Œuvr. posth., t. 2, 1783 (quoted from Devilliers), said that he had done the operation for empyema, and pus and gas issued from the wound.

The surgery of the chest in this particular may be summed up as by

32. Richter, who in 1795 (quoted from Sprengel) said that the cases where the chest should be opened are, besides empyema, hydrothorax, the effusions of blood and *emphysema*, effusions of drink, food or chyle into this cavity from lesions of the œsophagus or thoracic canal.

The findings of Hewson must have been considered important for Bell cites the following as a rival claimant for their credit.

33. Bromfield (quoted from John Bell).

Speaking of treatment of wounds of the chest, he said that in case an opening is made between the ribs, and a canula introduced whose diameter is larger than that of the wound in the lung, the air will be forced out as fast as it escapes from the lungs, the lungs will have room for their expansion and the danger of suffocation will be removed. " I remember a similar happy effect in accelerating the cure in a similar case by the patient playing on the hautboy which keeping the lungs forcibly distended for a long time together, kept the pleura closer to the ribs so that the union of the separated parts was most likely sooner effected than if he had trusted to the ordinary respirations."

Following Bromfield came Benjamin Bell, "who," John Bell says, "the most excusable of all, follows them gleaning up their mistakes."

34. Benjamin Bell. A System of Surgery, 1784-8. (The edition used was the VIIth, 1804.) Vol. 2, Chap. XXI, Section V, "Of air extravasated in the Thorax."

"Air collected in either of the cavities of the chest excites the same symptoms of oppression on the lungs and heart as those arising from water, blood, or matter."

"Collections of air may be produced in the thorax by different causes:

"1. As the process of putrefaction tends to extricate air from every part of the body in which it takes place, air may be collected in the thorax, from any of the organs contained in it being seized with mortification. This variety of the disease, however, will seldom fall under the care of the surgeon . . .

"2. Air may pass into one or both of the cavities of the chest from a rupture of the investing membrane of the lungs, and this again may occur from violent exertion in coughing, laughing, and crying, as well as from other causes.

"3. The surface of the lungs may be eroded by ulceration, or by purulent matter in contact with them becoming acrid, by which a passage may be given for air into one or other of these cavities.

"4. Wounds penetrating the lungs have sometimes produced collections of air in the chest; but in such instances the wound must be made by a small pointed instrument, pushed in an oblique direction. A wound produced by an instrument carried forward in a direct line into the lungs does not readily produce collections of air, as the air that escapes from the lungs passes out at the wound . . . but . . . oblique wounds . . . operate in the same manner as a valve, so that the extravasated air must necessarily collect in one or other of the cavities.

"5. The point of a fractured rib wounding the lungs is apt to induce collections of air in the thorax and a fracture of the sternum . . . or vertebra(e) may be attended with the same effect.

"These several causes may occasionally produce extravasated air in the thorax; but we meet with it more frequently from fractures of the ribs than from any of the others.

"The symptoms produced by air effused in the thorax differs only from those that occur from serum and purulent matter in their arriving more quickly to an alarming height. Instances have occurred of death . . . in a few hours from the fracture of a rib merely by air collecting in large quantities between the pleura and lung."

He then describes the symptoms and especially the development of emphysema.

"Till of late, patients in this situation almost constantly died . . . (but) of late years (the paracentesis advised by Dr. Monro) has been frequently performed and always with complete success."

In Vol. I, Chap. III, Bell discusses the symptoms by which the question

may be decided whether a wound has penetrated the chest cavity or not. He considers that those merely entering the pleural cavity are trivial compared with those wounding the lung. To determine this point "we should . . . cause the patient to make several full inspirations in order to force out any air from between the pleura and the lungs and at the end of each the contiguous skin should be so drawn over the wound as to prevent any more from finding access. In this manner nearly the whole will soon be discharged: when if we still find that air rushes forcibly out during inspiration we may with certainty conclude that the lungs are injured."

"In extensive wounds of the thorax where any part of the sternum or ribs have been removed, a portion of the lungs sometimes protrudes and does not readily recede . . . The protruding part should be replaced as soon as possible . . . unless it is in a state of mortification": in which case, excise.

This same writer (according to John Bell; I did not see the passage) advised for drawing off air from the thorax an exhausting syringe with an ivory or metal mouth of such shape as will allow it to be closely applied to the orifice of the pleura.

There is much that is interesting in this extract from Bell. Had he seen such cases of pneumothorax as he states were caused by putrefaction, or by coughing, laughing, and crying, or by ulceration of the lung? Was it the consensus of opinion of that age that such cases did occur, or was he stating every possibility which he could imagine? Hewson admitted the possibility that ulceration of the surface of the lung might be a cause but seemed to know very little about it. John Bell laughed at the idea that such acts as coughing, laughing and crying could rupture the lung, hence examples of these two groups must have been rare and disputed. Nevertheless, as will be seen later, all of these are now well recognized causes.

He shares the opinion of the others that it is not easy to produce pneumothorax by penetrating wounds of the chest. We wonder, however, if he really felt air come from the chest on deep inspiration. Many others also believed that inspiration did force the air from the pleural cavity, considering inspiration an active movement of the lungs and hence one which would tend to empty the chest cavity, but they must have mistaken the phase of respiration at which the jet did issue, or what is also possible, felt the air leave the emphysematous tissue around the wound, which possibly might occur since the tension of the skin is greater during inspiration.

35. **John Bell.** THE PRINCIPLES OF SURGERY. (The edition used was edited by Chas. Bell, 1826.)

The value of this writer's treatment of the subject consists in his very correct views of the mechanical principles involved. A few extracts (omitting those points which his predecessors also emphasized) are the following:

"Emphysema is less frequent in large wounds . . . When a rib is broken the point of the broken bone is pressed down upon the surface of the lungs and touches them; it abrades and lacerates the surface by the continual motion of the thorax, and it is often from the slightest and most superficial wound of this kind that the emphysematous tumor proceeds"—a laceration not deep enough to cause bloody sputum and which at autopsy can be found only by inflating the lung. But even though the abrasion be slight, "The air escapes at every inspiration; the air which is then within the cavity of the thorax is of course compressed so that at the next expiration this compression must force the air either back again into the lungs or out by the wound into the walls of the thorax . . . Thus at every inspiration there is a suction of some air which is drawn through the wounded lung, the air expands into the cavity of the thorax, the lung which gave out that air subsides again and lies almost entirely quiescent, partly from the wound in it which, like a rent in a bladder, prevents it being inflated, and partly from the oppression of the air within that cavity, where there should be a vacuum to cause its distension . . . Every new inspiration draws more air from the wounded lung, and every new expiration drives more air out into the cellular substance . . . The philosophy of this disease, if I may be allowed to say so bold a thing, is quite misunderstood; and it happens very strangely that the most palpable blunders are to be found in the writings of those who are looked up to on this point and who boast of their discoveries, and claim them as their own with an eagerness which would imply something very important . . ."

"The moment that the lungs are wounded they fall down and continue in this collapsed state until the wound heals, which it does in the course of a very few days. From that moment the use of the wounded lobe is lost . . . But this collapsed state of the lungs, which cannot be remedied, which must inevitably continue at least for a few days, while it is a cause of distress it is at the same time a chief means of safety . . .there is less blood thrown out into the cavity of the thorax to oppress the lungs . . . If the lungs when wounded were to continue in perpetual motion I do not know how we should expect a cure . . ." (for) "as the wounded lung lies in a collapsed state the edges of the wound are in contact with each other, . . . thus in two days the wound heals. . . .

"The blast of air from a wound in the thorax is often so strong that at every breath it will extinguish a candle and rushes out with considerable noise. This strong blast of air, so far from being a sign of wounded lungs, is often strongest when the lungs are absolutely entire; it is a sign of a free and open wound in the thorax . . . for whether the lungs be or be not wounded the air enters so freely by the outward wound that there

is no vacuum formed to give them play and therefore they fall down and lie collapsed till the outward wound heal. The thorax therefore has nothing to do with the lungs but is like a pair of bellows, having a large air-hole, which admits the air every time the breast rises; and when the breast falls again, that second motion blows it out . . . There is no vacuum to move the lungs. The lungs, whether they be entire or whether they be wounded, always lie collapsed . . . In a punctured wound there is no way for the blood or air to escape from the thorax, while at every stroke of respiration more and more air is drawn out from the lungs, till at last the blood and especially the air are so condensed that they not only oppress that side of the lungs but by hindering the free play of the diaphragm and loading the mediastinum they oppress also the other lung . . . and the patient dies. But in a free and open wound . . . we relieve not the oppressed and wounded side but the sound side of the lungs and all goes on tolerably well till the incision heal, and the function of the wounded lung is restored . . . .

"How impossible it must be to raise the wounded lung and to renew its function I think I shall now very easily explain, demonstrating to you at the same time that those who have intended this effect by their free incisions, had not founded their discoveries, as they prematurely called them, on good principles; for Bromfield writes very ignorantly in that chapter where he tries to claim the invention from Hewson. Hewson, again, is as vain of this trifle as if he were not rich in discoveries really honourable; and Mr. Benjamin Bell, the most excusable of all, follows them in the humble labour of gleaning and gathering up their mistakes . . .

"But the serious plain conclusion is this, that the lungs will continue giving out air till they heal; that while giving out air they must lie collapsed; that it is not desirable that they should be moved, for their lying unmoved is the best security against bleeding, and the surest way of making the lungs heal. And still further we cannot if we would raise the wounded lung, nor renew its function; it is rent so that it cannot be inflated; it is oppressed with air if the chest be close, or if the chest be open, the air passes in so freely that no vacuum can be formed to move the lungs; and as for inspiration emptying and expiration filling the breast, it could never have been spoken of but by a person who could talk about the lungs being burst with laughing. The incisions which have been so much commended are useful, not by raising the wounded lung, or renewing its function, for that attempt is absurd in all respects . . . (but because they) make[s] the wound lung collapse entirely, and set[s] the lung of the sound side quite free."

It will be seen that John Bell's explanation of the physics of pneumothorax is correct at nearly every point, while there are errors and inaccuracies in the explanations given by the three men whom he criticises so severely. Hewson, in his opinion, laid too much stress on the oppression of the wounded lung by the air, Bromfield sup-

posed the wounded lung could not expand because it was opposed. It is true that both seem to have ascribed to the lung an active part in inspiration and mistaken the phase of respiration which empties the chest. But Benjamin Bell was correct in supposing that strong expiratory movements, could fill the pleural cavity, and are perhaps the chief factor in distending a collapsed lung or bursting a normal one.

With the exception of this point, Bell (J.), has finished the chapter on the development of our knowledge of the mechanics involved in the development of pneumothorax.

These three men may have been wrong in a few particulars, but it should be borne in mind that John Bell does not challenge Hewson's claim to priority in the matter, he merely corrects his explanations; and to give a piece of work deserving such a careful criticism is much greater than to criticise it.

In conclusion then, the surgeons before Itard understood the condition of pneumothorax well; its mechanics were fairly well explained. They suggested it as a complication of medical troubles. Their views are more like the modern than are those of the clinicians following them.

The majority of writers on pneumothorax begin the bibliography of that disease with the thesis of Itard. Since this is the opinion of so many, especially the cotemporaries of Itard, his thesis must have been considered as marking a considerable advance in the knowledge of that disease, even the starting point. It is hard for us to see at first why they should have held this thesis in such high esteem. But this French student working for his degree, did open the whole subject for the medical profession. He showed that the condition was a not uncommon result of well-known, very common pulmonary diseases, and, what is no small point, he gave it the acceptable name "pneumothorax."

FROM ITARD TO THE PRESENT DAY.

36. Itard. DISSERTATION SUR LE PNEUMO-THORAX OU LES CONGESTIONS GAZEUSES QUI SE FORMENT DANS LA POITRINE. *Thesis*, Paris, 1803.

He reports 5 cases, three of his own, one of Selle, and one of Bayle.

CASE 1.—A man, 28 yrs. old, who practically starved himself in the hospital. At autopsy air was found in the pleural cavity, and the lung tuberculous.

Case 2.—Man, 26 yrs. old, who suffered three weeks from symptoms of suffocation; with the diagnosis of hydrothorax. On opening the chest very fetid gas rushed out. The diaphragm was distended into abdomen.

Case 3.—Man, 35 yrs., tuberculous, died after illness of six weeks. On opening right chest very fetid gas with odor of H_2S escaped.

Case 4.—Pneumothorax discovered at autopsy.

Case 5.—Notes given by Bayle. Man, 49 yrs., tuberculosis of some years' standing. Tympany even over heart present. At autopsy left-sided pneumothorax.

The conclusions he drew from the consideration of these two cases were;

"1. Pneumothorax is a disease of the chest consequent to latent pulmonary tuberculosis.

"2. It has for its immediate cause the long stagnation of a purulent exudate in a closed cavity; air is found following the absorption of this stagnant fluid and its decomposition to an aëriform fluid.

"3. The symptoms are: usually dyspnœa, more or less pronounced, and a tympanitic sound on percussion of the cavity where the gas is located.

"4. There are two varieties: (a) Effusion with progressive compression of the gas, pushing down the diaphragm, distending the side, pushing over the mediastinum, and accompanied by a suffocating dyspnœa—very rare cases. (b) Effusion without pronounced compression of the aëriform fluid, which occupies alone the space left vacant by the destroyed lung, and which is manifested after death by the emptiness of the thoracic cavity which contains little or no pus; 'a variety common enough as reasoning rather than observation forces us to admit,' in all cases where the diseased lung is replaced neither by pus nor by atmospheric air. Itard refers to cases in literature, Bennet's, Boret's, Morgagni's, and mentions several other similar conditions (gas in pericardium e. g.).

"5. Pneumothorax may be confounded because of the dyspnœa, dry cough, and distension of the thoracic cavity, with serous or purulent pleural exudates, from which it differs by the tympanitic note which characterizes all gaseous exudates. It may be confused with emphysema of the pericardium because of the identity of note, and with tuberculous cavity of the lung, but differs from the first by difference in position, and the note on percussion, and from the second by the absence of the purulent expectoration.

"Pneumothorax presents only negative signs, and if it be permitted to call the attention of practitioners to it, it is much less to concern them with its treatment as to guard them from the mistakes into which it may lead them."

To sum up Itard's views we may say: He found it only on the autopsy table but refers to it as a condition to be recognized during life; he demonstrated its relation to tuberculosis; he considers the process to be as follows, the lung is destroyed by a chronic suppurative disease, and its space taken by the products of the suppuration,

and an aëriform fluid. Tuberculosis is the only causative disease mentioned. The origin of this aëriform fluid he cannot satisfactorily explain and objects to theory; he suggests the decomposition of the fluid as the source. One thing is certain, it was not, in his opinion, atmospheric air, and the odor of H_2S would seem to justify this conclusion. Herein lies a new element in knowledge of the disease.

The following quotation from Bayle is given to show the justification of Laennec's claim to be the first to recognize pneumothorax during life. He says of Bayle's treatise, that "It is the most accurate and full of any that have been written on diseases of the chest. It contained five cases of pneumothorax with serous or purulent effusion—no one was suspected during life, and in two, air was not discovered after death, although its existence appears clearly from the dissection." (Cases 11, 40, 42, 43 and 45). Bayle was a most accurate observer "but general symptoms and percussion alone will not reveal a pneumothorax." (Laennec, loc. cit. p. 480.) We give one case as an example:

37. Bayle. Recherches sur la Phthisie pulmonaire, 1810, page 176.

Case 11.—A man, 21 yrs. old, a history of tuberculosis of 4½ months' duration, died September 23, 1803. The left side resounded well, there was dulness on the right side. At autopsy much gas escaped from the left side with some explosion. The cavity in that chest would hold three pints but contained only two ounces of fluid. Bayle explains that the serous fluid was absorbed and replaced by aëriform fluid, which was not rare and refers the reader to Itard's thesis.

38. Boyer. Traité des mal. chirurg., 1814, Vol. III.

This surgeon is mentioned chiefly because of his eminence, and the fact that although a professor at the university from which Itard was graduated, and writing after the appearance of Itard's thesis, he does not mention it, nor say anything which would indicate that the prevailing idea concerning air in the pleural cavity as a result of injury had at all changed. This it seems to us is strong evidence in favor of the view that at first the pneumothorax of Itard was considered an entirely different condition from that following injuries of the chest.

In Volume VII, dated 1824, he lays considerable stress on the diagnosis of penetrating wounds. One point he mentions (as of little value) is to close the patient's nose and mouth and expire (showing that he knew the mechanics of respiration) forcibly to feel if any air escaped from the wound. He calls attention to the fact that emphysema at the wound

almost always means a wound of the lungs, thus differing from Petit. Five cases of hernia of the lungs are cited (Hildan, Tulpius, Ruysch, Roland, Loyseau) and several of emphysema after wounds.

The thesis of Itard did not attract much attention until the appearance of Laennec's work. The following article is important since it is a careful review of the whole subject, in a famous French encyclopedia of medicine, and written between the works of Itard and Laennec.

39. Rullier. EMPYÈME. *Dict. des Sciences médicales*, Vol. 12, 1815.

Rullier defines empyema as a collection in the chest of pus, blood or serum, air or gas. It is not an independent disease but always secondary.

P. 133. An empyema of air may follow:

1. Wounds of the lung.

2. Superficial ulceration of the lung (e. g., after empyema of pus).

In all the above cases it is atmospheric air, and forms one part of a composite pneumothorax of air and a fluid.

3. In other cases the gas is a stranger to the air and evidently results from the putrid decomposition of exudates. "We have seen in the anatomical society an enormous pneumothorax which appeared due to a cause of this sort, and M. Itard has furnished several other remarkable examples of it. One should, however, be skeptical concerning these cases, lest such a gaseous effusion into the chest proves for the most part the product of a kind of adventitious secretion of the pleura itself."

By this author therefore Itard's cases are only good examples of a formerly recognized form of empyema of gas, but he more than Itard has the gaseous secretion obsession.

The article is long and exhaustive and contains an excellent review and bibliography of empyema of all forms.

40. Piorry. PNEUMATOSE. *Dict. des Sciences médicales*, 1815, Vol. 43, page 342.

By pneumatosis the writer means the condition in which gases occur normally or pathologically in the organs. This may be (1) at the surface of the skin, (2) in the great cavities of the body, (3) in the vascular cavities, (4) in the parenchyma itself. By (1) the cutaneous respiration is meant. Under (2) are the cavities lined by (a) mucous membrane, and (b) by serous membranes, etc. In an article of 28 pages the opinions on all these occurrences are given.

(Frank seems to be the chief exponent of this disease, the belief in which gave pneumothorax much more interest than it would otherwise have received.)

The most important contribution on pneumothorax was made by Laennec. He insists rightly that the presence of this disease during

life could be detected with certainty only with his newly invented stethoscope and that to him is due the credit of bringing a disease formerly, he says, recognized only on the autopsy table, within the scope of the clinician.

41. Laennec. De l'auscultation médiate, 1819.

(Forbes' translation of the III edition, 1831, was used, as question of priority need not be considered, but the date 1819, is used in locating the position of this article since the first edition appeared that year.)

The author briefly reviews the *history* of the question. The disease was not rare, but overlooked; all that can be found are a few examples imperfectly described. Anatomists and surgeons had found gas under high tension on the autopsy table and while operating for empyema. The best memoir is by Itard. This briefly is his historical summary:

Laennec *classifies* cases in Chap. IV of pneumo-thorax as follows:

1. Cases in which the gas arises from decomposition of an effusion, in tuberculosis. This kind, which Itard described, is "pretty common."

2. Due to rupture of a tubercle or vomica simultaneously into pleura and bronchus, and the gas is atmospheric air. "I consider this the commonest of all. The air possibly causes the pleurisy and not vice versa as in Itard's cases, yet the vomica may rupture into the pleura alone, and this cause the pleurisy, and this by decomposition the gas. Pneumothorax may accompany hydrothorax. Bayle saw one such case, Laennec none, although he frequently saw a certain amount of air with the serum in the symptomatic hydrothorax supervening just before death.

3. Gangrenous eschar of lung softened and evacuated into pleura, and gas formed from decomposition.

4. Gangrene of pleura.

5. Decomposition of blood. Littré's case (q. v.) is his illustration of this group.

6. Rupture of pleura from external violence. Hewson's case cited.

7. Rupture of air cells in emphysema and extravasation of air under pleura rupturing this membrane. He thinks he has seen this form.

8. Even in acute pleurisy at its commencement and without decomposition of the effused fluid there may exist a gaseous effusion.

9. The formation of an aëriform fluid in the chest. The gas is inodorous, there is no solution of continuity, no other effusion or any perceptible change of structure. "I have often perceived the escape of an inodorous gas on opening the pleura without perceptible affection of the pleura"—although it may be drier than normal, and in one case was parchment-like. "Slight rupture may be imagined, but probably not without external violence, and we know idiopathic formation of air or secretion does occur in the body," viz.: pericardium, synovial capsules, under the arachnoid, etc.

"It would appear that air or an aëriform fluid exists naturally in small quantities in the pleura." "M. Ribes says in opening serous cavities of dogs a small amount of air always escapes; this may probably, however, be merely the natural serous exhalation in state of vapor." Whatever the nature of the gas in simple pneumothorax it may long remain without giving rise to any inflammation of the pleura, as would no doubt be the effect of atmospheric air introduced by a tuberculous excavation communicating simultaneously with the bronchi and pleura, since air secreted by the vessels of the pleura must be animalized, and therefore "less likely to irritate than a body so thoroughly extraneous to the animal system as the air of the atmosphere."

Case 35.—To show that the air secreted may not cause severe pleurisy. Man 65 yrs. of age; cough of two years' duration; died suddenly from severe pain in the abdomen. At autopsy the right chest was found distended, giving a louder sound on percussion than normal. An inodorous gas escaped under pressure from this chest. The lung was small, the pleura drier than normal, no fluid was present. A space was left by air which would fill about two pints. The upper lobe was adherent covered by a cartilaginous incrustation of the size of the palm of the hand, under this a cavity the size of a small orange communicating with the pleura by a small opening. Other lung tuberculous. He is in doubt as to the origin of the air, but is sure at least it did not cause pleurisy.

Symptoms and Signs of Pneumothorax.—(Several sections here combined.)

Symptoms.—The symptoms are obscure since they may belong to many other affections. The only one which is pretty constant is a certain degree of *dyspnœa*. *Cough* may be absent.

Exploration of the chest by succussion. He reviews its history. Laennec first insisted that air and fluid must be present. If very little fluid, one may need the stethoscope; if more, the naked ear is sufficient; sometimes one can hear at a distance. Even the hand can perceive the motion of the fluid during alternate stooping and raising of the chest. "The Hippocratic fluctuation is among the few signs which of themselves convey to the least experienced observer a full and entire conviction of the existence of the disease." Nevertheless one may be deceived. He was once in a case with a very large tuberculous cavity of the lower two-thirds of the right lung, which was everywhere adherent, and hence was really the wall of a cyst, being only one or two lines thick and at one point was represented alone by the pleura. One must exclude fluctuation arising from the stomach after drinking. Morgagni thought succussion too severe, but Laennec says it causes very little discomfort if well done. Don't shake the body much, only the shoulders with a quick motion, and stop suddenly.

Inspection alone is no criterion. The affected side may be larger or smaller than the normal. Smaller from absorption of a part of the air and liquid, and if distended may appear the more normal of the two sides.

PERCUSSION alone gives no certain diagnosis. If much air is present one gets a clearer tone than normal, but this even when very distinct leads to error, for one may select the good side (since duller) as the diseased one. If fluid also be present the sides may sound equally clear.

Distension and tympany together more often deceive than aid.

In Corvisart's clinic Laennec saw at autopsy many cases of pneumothorax which were not suspected during life, and Corvisart's ability as diagnostician is not to be questioned. It shows the insufficiency of observation and percussion in diagnosis. We must add to these MEDIATE AUSCULTATION. "Whenever we find one side of the chest sounding more distinctly than the other, and at the same time perceive the respiration very well in the less sonorous side and not at all on the other, we may be assured that there exists pneumothorax on the latter. We may be equally sure of our diagnosis when both sides are alike sonorous, and even although the afflicted side were somewhat less sonorous than the sound one. . . . Air being a worse conductor of sounds than liquids, it is more difficult to perceive the respiratory sound of the healthy side on that which is diseased in pneumothorax than in empyema."

The *metallic tinklings* are so intimately associated with Laennec's name and have been the cause of so much dispute that we will here give Laennec's full description of this physical sign.

They are "a peculiar sound which bears a striking resemblance to that emitted by a cup of metal, glass or porcelain when gently struck with a pin, or into which a grain of sand is dropped. . . . it is perceived during respiration, speaking and coughing, but is much more perceptible during the latter two than the former. The reverse of this is, however, sometimes the case. It is in general heard in a most striking manner during coughing, and when in any degree doubtful, this action ought to be performed." He describes it again as a "slight, sharp sound like that occasioned by the vibration of a metallic cord touched by the finger." "The metallic tinkling always originates in a morbid excavation within the chest, containing partly air and partly liquid. It exists only, therefore, in two cases, viz.: where a serous or purulent effusion coexists with pneumothorax, or when a large tuberculous excavation of the lung is only partly filled with very liquid pus. It is further necessary for the manifestation of this phenomenon, in cases of empyema or hydrothorax complicated with pneumothorax, that the cavity of the pleura should communicate directly with a bronchial tube by means of a fistula, such as has place when a tuberculous vomica abscess or eschar of the lungs opens into the chest. This sign may on this account be considered as pathognomonic of this triple lesion." Laennec considers these tinklings an aid in estimating the size of the fistula and the relative amounts of air and fluid in the chest, since they are "more distinct according as the fistula is larger, while the extent of the vibrations of the sound corresponds with the extent of the spaces occupied by the air." "Sometimes the tinkling assumes another character and strikingly resembles the sound produced by blowing into a flask or bottle." He named the latter "*utricular buzzing*" or "*amphoric*

resonance." (Forbes suggests these as translations of Laennec's term "bourdonnement amphorique.") It may coexist with or alternate with metallic tinkling. In a case of empyema one month after operation he was able to produce these phenomena; the metallic tinklings by injecting fluid into the pleura through the wound, in such a way that it fell on the fluid in the cavity, while the utricular buzzing was produced by the air entering the chest through the wound. By diminishing the size of the hole if the patient spoke a distinct tinkling was heard. He therefore concluded "that a large communication with the external air converts the tinkling into simple buzzing."

The metallic tinkling is scarcely ever found in simple hydropneumothorax (i. e. with no fistula); here neither respiration, voice nor cough produce it. But it may be caused by the patient suddenly raising himself in bed, and as a drop of fluid falls from the upper part of the cavity of the pleura into the fluid beneath, it produces a sound like a drop of water let fall into a flask three parts empty, and this sound is immediately followed by a very distinct metallic tinkling of longer duration than that produced in another manner. A similar sound is produced after a drink of water by its fall into the stomach.

But if rare in simple hydropneumothorax it is constantly observed during coughing, speaking or breathing in the case of a fistulous communication between bronchi and pleura, or at least the utricular buzzing is so if the tinkling is not fully developed, "and indeed these are the only signs which enable us to recognize the fistulous communications in question." "The extent over which the metallic tinkling and buzzing are perceived together with the Hippocratic succussion serve to discriminate the case from a vast tuberculous excavation. The Hippocratic succussion no doubt of itself demonstrates the nature of the affection and even here the other sign is highly important; and we cannot be too well assured of the existence of a disease so severe as this and which has hitherto perhaps never been recognized in the living subject. This may seem a bold assertion, but I am well assured of its correctness."

Movable dulness.—(A sign upon which so much dependence is now laid, we would have expected so accurate a physical diagnostician as Laennec to have valued.) Percussion he says would only aid in the diagnosis of pneumothorax if the chest were percussed when in different positions. "In these circumstances the gas rising always to the part of the cavity which is superior, the sphere of the dead sound will vary with each posture of the patient;" but mistakes caused by adhesions of the lungs and the inconvenience to the physician and patient will prevent its use except where the nature of the trouble is already suspected. (It would be of interest to know his opinion of its value in pleurisy.) Under the signs for acute pleurisy he says: "To exclude pneumonia I have seen physicians try to get a mark of distinction between the two diseases by placing the patient in different positions, and I have myself made a like experiment but without any satisfactory result. This might be expected since the

chest is always full and fluids change place by position only in a vessel that is more or less empty. In the chest the fluid can only change its position by compressing the lung." (It is not strange that one who sees no value in movable dulness as a sign of pleural effusion, and if he percussed as he was wont with his stethoscope he may well have missed it, should underestimate it in pneumothorax, but it is stranger that one who recommends succussion should condemn trying movable dulness because it was inconvenient to *physician and patient.*)

Double Pneumothorax.—On page 487 of this edition Laennec says that it is by no means uncommon to perceive the escape of a very small amount of air discovered by the hissing sound, from each side of the pleura on opening the chest of a dead body. Accumulations of air of this kind are commonly small in amount and are usually combined with a small amount of fluid, and must be considered as a product of changes immediately preceding death. But he adds, double pneumothorax occurs under other circumstances but is extremely rare. He gives notes on two evidently genuine cases. (The air he often heard hissing in the vicinity of a chest on opening it may have rushed into the chest rather than out.)

Diagnosis of Pneumothorax.—Page 484.

Laennec divides them into three groups:

1. Simple pneumothorax. Here the percussion note is good, even preternaturally sonorous; respiration not at all perceived.

2. Pneumothorax with liquid effusion, where we find the above signs, with in addition dulness at the base varying with the position of the patient, and the succussion splash.

3. Pneumothorax with effusion and open fistula between pleura and bronchus. Here we find all the signs of 2, and in addition metallic tinkling or amphoric resonance which commonly alternate.

The first two varieties cannot be confounded with any other affection. The third could be with a vast tuberculous cavity nearly empty; but there is little danger of mistake; in the latter is some remains of pectoriloquy, the sphere of the tinkling, amphoric resonance, and tympany is very circumscribed, and there is no splash on succussion. The cough sometimes causes slight fluctuation (heard with stethoscope) but never in pneumothorax.

One must exclude emphysema, which is easy, since in this there is never loss of respiratory sound generally, nor its comparative integrity at the root of the lungs.

"The effusion of air comes on suddenly and cannot exist for any length of time without giving rise to severe symptoms and even causing death. I have never seen pneumothorax in any person who was not confined to bed."

In pleurisy there is lack of succussion splash and of metallic tinkling and amphoric buzzing. If a pneumothorax supervenes on a pleurisy the percussion sound gets clearer.

Treatment.—The exact diagnosis of pneumothorax and of each variety is purely a matter of speculative knowledge or useful only in the prog-

nosis. Simple pneumothorax are the cases most successfully treated by puncture, but he considers simple pneumothorax as of no great severity, since the gas is more readily absorbed than the liquid effusion. He defends this conclusion by mentioning the frequent gaseous effusions in other situations (pericardium and joints, especially the knee), "which disappear spontaneously and frequently in the course of a few days or even hours." Pneumothorax with liquid effusions and especially with a fistula are more serious, yet a cure is not impossible, even in the severest cases. Operation is recommended only when there is imminent danger of suffocation or rapidly increasing emaciation and debility. "In every other case (unless the other lung gives no indication of tuberculosis) I think we should content ourselves with supporting the patient's strength, promoting absorption, . . . and by a regimen regulated according to the digestive functions, neither too rigid nor too analeptic."

The value of Laennec's opinion concerning this disease cannot be appreciated unless the cases of which he gives one or more to illustrate each of his points be carefully studied.

One is impressed with the number of cases he saw, of which he gives us careful notes on twelve. But he assures us that these are only selected cases and that he could have multiplied the examples. For instance he says that he saw at least thirty (!) cases similar to one illustration. A few very much abbreviated protocols are here given.

Case 40.—Pneumothorax. Subacute Pleurisy. Tuberculosis.—Man of 29 yrs., ill with tuberculosis four months. Pneumothorax developed quite suddenly. When this patient sat up, "I heard a sound like that produced by a drop of liquid let fall into a flask containing a very small amount of water, and this sound was followed by a second, by a tinkling such as is occasioned by striking a glass with a pin. Neither the voice, cough, nor the respiration was attended by any sound of the same kind." Hippocratic succussion splash heard. Diagnosis—Left-sided pneumothorax with small effusion, but not developing from the rupture of a softened tubercle, since no metallic tinkling was heard except as above.

Case 11.—Chronic Pleurisy. Pneumothorax. Partial Gangrene of the Lung.—Man of 22 yrs. Tuberculosis for 3 mos., then pleuropneumonia of right side. Pneumothorax accidentally discovered from hearing the metallic tinkling, and the succussion splash. "Nor did the liver appear pressed down into the abdomen." The diseased side was even smaller (due to the former pleurisy). The empyema was operated for, and the patient died in four hours, since the incision made in the fifth interspace of the mid-axilla opened the abdomen, the diaphragm being adherent.

Case 36.—He considers that in this case gas was exhaled as the fluid receded. It was a case of pneumothorax following pleurisy. The patient (a man of 36 yrs.) gave a tuberculous history. The pleurisy was discovered one day; it was on left side and purely latent. Later percussion

became natural over the area where formerly was ægophony, but no respiration at all was heard, except over lower two-thirds, where were still ægophony and dulness. There was no dilatation, no metallic tinkling, no succussion splash (tried), hence Laennec diagnoses, simple exhalation of gas into the pleura, no fistula. Recovery. (Such a case Proust maintains, and rightly, is not pneumothorax, but simple pleurisy. It is of interest that in no case did he emphasize the dislocation of the heart.)

But many of Laennec's cases were probably not pneumothorax, but simple pleural effusion, and contrary to his statement his two first groups can very easily be confounded with some other diseases (v. Proust).

Laennec's contribution to the subject is important and interesting. Marais said of him " Since Laennec, one has said nothing, it is necessary to admit, which makes easier the recognition of pneumothorax. He was one of those men who go to the bottom of the subject with which they deal, and leave little behind for others to do in that subject. But if he said the last word concerning the existence of the diagnostic signs of the disease he did not succeed so well in their interpretation." He claims to have been the first to recognize the existence of the disease in the living subject, and it was thanks to his stethoscope that he did so. But mediate auscultation was an obsession with him and he belittles the data given by the clinical history, the eye and the hand. His classification of the cases according to their etiology is fair, according to physical signs, faulty. His essential pneumothorax cases have all been long ago condemned, and the ease which he ascribes to the body to secrete gas, questioned and denied. The air hissing out of so many normal chests may have hissed in, the water vapor seen to rise from a warm organ may have deceived him. He is the first since the Hippocratic school to give succussion its true value, and he described the most of the auditory signs although their explanation is still in dispute.

42. Piorry. Pneumothorax. *Dict. des Scien. méd.*, 1820, Vol. 43. (This review of the subject is important as giving the view of an eminent clinician on the work of his cotemporaries who first brought this disease into prominence.) Piorry is concerned that the disease shall be correctly named, and thinks pneumato-thorax more correct than the common name pneumothorax. His historical review is good, mentioning the work of Haller, and stating that Bichat and Roux, as well as Laennec, believed in the presence of an elastic fluid in the chest normally.

The same factors governing the entrance of fluid into the peritoneum operate also in the chest. Gas in the chest may arise from:

1. A communication between the pleura and bronchi or between the pleura and the external air.
2. Gaseous exhalation.
3. Inflammation.
4. Putrefaction of a serous effusion.
5. From a gangrenous slough at the expense of the lung.

Among those who had noticed such cases were Ponteau, Littré, Riolan, Portal (who used candle flame as proof that air issued from chest), Houllier, Selle, Bartholin, Baillon, but they paid no attention to the condition till Itard's thesis. He gives Laennec due credit for his work.

Pneumato-thorax is more common than is reported, and almost always a symptom of a grave disease. Laennec believed in an independent disease, but gives but one case in which the source of the gas is uncertain. We may ask if here there was not small loss of substance of the lung or pleura, but however that may be, analogy teaches that there is a "pneumatosis," the result of more gaseous secretion than is normal, but whether symptomatic or essential, pneumothorax is important.

Symptoms.—Only since Auenbrugger can we diagnose diseases of the chest. Corvisart improved percussion but admitted it was imperfect in pneumothorax. Piorry advises to use *movable dulness* as a diagnostic sign and differs from Laennec concerning its expediency.

Bichat's sign that pressure on the abdomen is much more painful when air or fluid are in the chest than when inflammation, and which Laennec pronounces of little value, Piorry says is excellent and harmless. Distension of the intercostals is important only in extreme cases. The introduction of succussion, a test "shrouded in the night of time," is due to Laennec, to whom also he gives full credit for the auscultatory symptoms.

In the opinion of this writer therefore Itard and Laennec deserve all the credit usually granted them. He considers also that pneumothorax is something more than a condition of air in the pleural cavity, it may be an independent disease.

A very careful piece of work on the gas in the pleural cavity in cases of this disease, as well as the first, was that of

43. Davy (J.). Observations on Air found in the Pleura in Pneumothorax, with Experiments on the Absorption of various kinds of Air introduced into the Pleura. *Philos. Trans.*, 1823, p. 496.

A soldier, 30 yrs. old, admitted for phthisis pulmonalis, died, and at autopsy pneumothorax was suspected, since the diaphragm projected down into the abdominal cavity. The chest was therefore opened under water and the gas collected (212 cu. inches). By inflation of the lung the fistula was easily found. "On examining minutely the communica-

tion between the cavity of the chest and the *lung a kind of valvular structure was discovered*, which would allow of air being pumped into the pleura in the act of inspiration, but not of its escape in expiration, owing to which no doubt the accumulation of air in question took place. Even at the risque of being tedious I must attempt to convey some idea of this structure. Between the false membrane of the vomica and the pleura there was a small irregular sinus not exceeding an inch in diameter, the sides of which, though not adhering of course, were in contact or very nearly so. This sinus was the channel of communication and contained the valvular structure alluded to. It opened into the cavity of the chest by a hole in the pleura pulmonalis about the size of a crow quill, and into the vomica by three smaller holes in the substance of the lung, not corresponding with the former, so that a probe could not be passed from one into the other in a straight line, and consequently when the surfaces of the sinus were pressed together by the compression of the air in the pleura in the act of expiration, the communication through which the air entered was closed and its exit prevented."

The gas obtained was analyzed, lime water being used to absorb the CO_2 and phosphorus the O.

100 parts contained CO_2 8 parts.
N 92

(The autopsy was made fourteen hours post mortem.)

To learn how the O had disappeared, air was injected into the pleural cavity of a dog removed in forty-eight hours and 8 cu. in. obtained.

N = 93 parts.
O = 7

hence the O is absorbed faster than N.

30 cu. in of gas (80 pts. N, 20 CO_2) were injected into the right pleura of a dog, and two days later a similar amount of gas (N 63.2, O 16.8, CO_2 20) into the left side. In 24 hrs. the dog was killed.

Right side CO_2 18.3 parts.
N 78.3
O 3.4

Hence in three days O is absorbed faster than CO_2 and this than N. The gas from the left side contained CO_2 25 pts.

N 70.6
O 4.4

Hence we can say that in the case of pneumothorax examined the CO_2 was derived not from secretion or exhalation from the surface of the pleura but from the respired air through the ulcerated opening.

Davy continues to study this selective power of the pleura to absorb gases.

Into the left pleura of a dog 20 cu. in of a gas (CO_2 = 57.5, H = 42.5) were injected; two days later 30 cu. in. (N 44.5 pts., nitrous gas 55.5). The dog died in five hours. In the gas from the left side the H had evidently disappeared (since after removing CO_2 with lime water the remaining gas was not inflammable, hence was chiefly N). The gas

from the right side was treated with green sulphate of iron, and there was found N 93.1 pts., nitrous gas 6.9 pts.

The pleuræ were normal but the right lung contained blood and serum.

The conclusions from these observations were, that most of the CO_2 and all the H were absorbed and considerable N appeared; that all of the nitrous gas was absorbed, but it caused death, although no alteration of the pleura. This work on H was repeated and also the nitrous gas did not cause death if the other lung was good.

What is the source of the N? Did it exist previously in the pleural cavity? He quotes Laennec, Haller and Hamberger on this point. Davy by repeated experiments, opening dogs' chest under water, convinced himself that this is not true. He then examines the point whether or not it is derived from the blood as an exhalation or secretion, and although his experiments he says are not conclusive, yet he thinks that to be the case.

He tried nitrous oxide once and found it completely absorbed. A mixture of air and the gas were introduced and in 24 hrs. the remaining gas contained O 10 pts., N 90 pts.

44. Davy. *Philos. Trans.*, 1823.

A soldier, discharged from the army for hæmoptysis succeeding a severe fall on the left side of the chest received eighteen months previously, showed suddenly after a severe spell of coughing the symptoms of pneumothorax. "The left side was found more protuberant and in all its dimensions larger than the right. It was tense and on percussion sounded hollow rather than tympanitic . . . the heart was found beating under the right mammilla." A trochar with an empty bladder attached was thrust into the chest. The air rushed out, distending the bladder, which was then removed. The air continued to rush from the chest as from a blow-pipe. The relief was immediate and great and in time the patient discharged.

Gas obtained, 25 cu. in., N 93 pts., CO_2 7 pts.

Davy. *Philos. Trans.*, 1824, Mar. 4.

The same case again above mentioned is again reported. One month after the previous report symptoms of hydrothorax supervened. When shaken the splash could be heard at several yards. The operation advised by Hippocrates was performed, a hole being bored through a rib with an auger. At intervals the gas was analyzed.

July 15	CO_2 7.5 pts.	O 2.5 pts.	N 90 pts.
July 20	" 6	" 5.5	" 88.5
July 29	" 8	" 4	" 88

The patient died. The gas removed at autopsy twelve hours P. M., CO_2 16 pts., O 1.5 pts., N 82.5 pts.

Two perforations were found by inflating the lung. He tries to explain the reason for the differences in the above analyses, but decides it lies in the absorbing power of the pleura.

The following thesis of a French student, evidently one of Laennec's, since the latter's name is among those of the faculty of Paris given in this thesis, may perhaps be considered as expressing Laennec's opinions.

45. Venot. Considerations sur le Pneumothorax. *Thesis*, Paris, 1824.

He begins by sharply distinguishing between the secondary pneumothorax of Itard and the primary of Laennec, and proceeds to claim the existence of the latter as a truly independent disease. Essential pneumothorax is not, he says, demonstrable by immediate observations of cases, but nevertheless one should regard the condition as primary. One reason for this opinion he finds in comparative anatomy, the groups of cysts containing gas attached to the mesentery and intestine of hogs.

Yet he admits pneumothorax is more often secondary. He relegates Itard's explanation of the presence of gas to the "ancient school" and out of harmony with the known laws of physics of chemistry (the absorption of a stagnating exudate and its decomposition to an aëriform fluid). This could be true only for gangrene cases. To explain these cases of idiopathic pneumothorax one need not assume air-conveying vessels or other anatomical subtilties, but only an abnormal secretion. He cites emphysema as such a case. Gaseous exudates in the pleura are permanent gases of unknown composition.

He then cites four cases of idiopathic pneumothorax (which Proust,[124] characterizes as pneumothorax in name only.)

The writer is evidently defending Laennec's views and is lauding him at the expense of Itard. The one object of the paper is evidently to establish more firmly the right of idiopathic pneumothorax to consideration.

This same year another student presented a thesis at Paris on this same subject.

46. Clemanceau. Dissertation sur le pneumothorax. Paris, 1824.

This thesis contains an excellent review of the subject with many references to literature, among which are those to Bell, Boyer and other surgeons. The writer presents nothing new in the way of cases or experiments. Laennec's views are emphasized.

47. Brierre de Boisemont. Recherches sur les Pneumatoses. Paris, 1825.

This thesis, on the title page of which, among the faculty of Paris, Laennec's name appears, and in which the references to Laennec's opinion are very complimentary, may perhaps express Laennec's opinion on the above subject. In conclusion he says that under certain conditions the organs which normally produce gas produce an abnormal amount, or gas of a different composition; sometimes those organs not normally pro-

ducing gas do so in abundance. For instance, the digestive organs may be distended with rapidity by gases which can only be formed by exhalation, e. g. in an hysterical woman. In the case of serous cavities the gas is secreted owing to a local or general disease.

One of the most famous names of this time was that of

48. Louis. RECHERCHES ANATOMICO-PATHOLOGIQUES SUR LA PHTHISIE. 1825. Translated by Cowan, 1836.

Louis evidently did not think pneumothorax such an important disease, or at least did not admit it. He very seldom used the term pneumothorax and gives to the chapter containing his observations on this condition the heading, "Symptoms of Perforation of the Lung by a Softened Tubercle discharging itself into the Pleural Cavity." He evidently corrects his diagnosis on some cases previously reported by him and reprints them, recognizing that pneumothorax must have been present. Metallic tinkling he has heard in large tuberculous cavities which communicate with the bronchus as well as in pneumothorax, but the distinction between the two cases is easy, for in the former case it is accompanied by pectoriloquy and tracheal respiration, both of which are absent in the latter. He gives a good case to illustrate this point. A girl 20 yrs. old, suffering from phthisis—percussion was clear under both clavicles, pectoriloquy and metallic tinkling during the cough on inspiration and expiration heard under the right clavicle. The right lung showed "an excavation occupying three-fourths or four-fifths of the total volume of the lung." "The clearness of percussion below the right clavicle combined with the pectoriloquy and the metallic tinkling in the same region proved to demonstrate the existence of a vast excavation containing both air and liquid."

OBSERVATION 38.—Man 36 yrs. old. "Three days before entering the hospital he was attacked suddenly after having vomited on account of being exposed to the fumes of burning charcoal, with a violent pain in the left side, accompanied with dyspnœa and great anxiety." Two days later the percussion note was unusually clear over the left side; no respiratory murmur, no metallic tinkling; the intercostal spaces were prominent and wide. About three weeks later metallic tinkling was heard. The chest on the left side contained four quarts of pus surmounted by a small amount of air.

OBSERVATION 39.—Woman, 45 yrs. old. Onset was with a sudden attack of pain near the inferior angle of the scapula, at first moderate, then suddenly very violent, with dyspnœa, feeling of anxiety, continual cough, and orthopnœa. Percussion much clearer on the right side, respiratory murmur absent except in upper back; no metallic tinkling. Death. On opening chest gas escaped, and lung occupied less than one-third of the pleural cavity.

OBSERVATION 40.—Woman, 32 yrs., attacked by a violent and sudden pain along the vertebral column accompanied with dyspnœa and anxiety.

Percussion was clearer on the left. No respiratory murmur was heard, only mucous râles which appeared to traverse an empty space before reaching the ear. There was no metallic tinkling. At autopsy a small quantity of gas escaped from the left side. This lung contained a tuberculous cavity with a perforation the size of a small pea.

Observation 41.—Woman, 26 yrs. old. "In the night of the first of January she experienced suddenly a sensation in the left side of the chest similar to that which would have been caused by the circulation of a gas from below upwards in this same part; the breathing at the same time became very difficult." On the left side the chest resounded like a drum—no respiratory murmur nor metallic tinkling could be heard; the aphonia was complete. Later metallic tinkling was heard. Percussion note of left side was clear. This side was found to contain rather more than 100 cc. of a gas which proved to be CO_2.

Cases 42 and 43 are similar (page 402). "So that whenever in a case of phthisis a violent pain on one side of the chest shall come on suddenly, accompanied with dyspnœa and anxiety, and with all the general symptoms of acute pleurisy, we may conclude that perforation of the lung has taken place." Again he says: "In the absence of pain we did not hesitate about the existence of perforation of the lungs when the patient told us of the sudden invasion of dyspnœa and anxiety." However, he admits that the diagnosis is assisted by percussion and auscultation. He quotes Laennec concerning the physical signs. Metallic tinkling is present when fluid is present and the fistula open. But if the fistula is closed, or no or too little fluid present, there will be none. "Auscultation cannot therefore decide perforation in any case until some time after its occurrence; and in those instances where no communication with the pleural cavity exists the diagnosis depends entirely on the other symptoms." The quantity of fluid necessary for the production of the metallic tinkling does not seem to be very considerable, since he has heard it when percussion detected no dulness of the sound. On the other hand a large accumulation of fluid does not appear to interfere with its presence.

"The seven preceding observations are the only examples we have met with, and in all the rational symptoms independent of auscultation and percussion were sufficient for the diagnosis of the lesion. This seems, however, not to be always the case, for in the three examples related by Laennec . . . these symptoms do not appear to have been present. When this is the case nothing would lead us to detect its existence, and if the discovery were made it would be by accident, as it were, by auscultation and percussion, and this could happen only where there is a communication existing with the bronchia, for then alone is there metallic tinkling." "Percussion and auscultation would also be the only means of diagnosis which we should have of perforation of the lung if the symptoms were but slightly marked, since they would indicate a pneumothorax of which perforation of the lung is incomparably the most frequent cause."

A writer cannot be judged from one book nor indeed by the productions of one period of his life, hence we do not know but that later Louis came to respect pneumothorax more than at this the thirty-ninth year of his life. But at this time he seems to have considered it more as an incident in the course of a case of phthisis, and scarcely worthy of a name. It was the result of the rupture of a softened tubercle into the pleura, and that is all. This clinician it will be seen lays especial stress on the subjective symptom-complex; auscultation and percussion help. The succussion splash he does not mention. But the diagnosis of air in the pleura is not as easy as this. There is some doubt that all of Louis' cases were pneumothorax at all, for sudden agonizing pain in the chest followed by dyspnœa does not always mean a rupture of the lung, and the tympany and amphoric breathing may exist over a solid lung, a collapsed lung, or a fluid, under certain circumstances. The truth of this was not apparent, however, until the literature had been well stocked with "interesting cases of pneumothorax."

49. Alexander. Case of Pneumothorax. *Edinb. M. and S. Journal*, 1826, XXV, p. 294.

This case was that of a man 22 yrs. old, in whom pneumothorax developed during the last stages of consumption. The diaphragm was found projecting convex downwards, pushing the right lobe of the liver directly over the spine. The pleural cavity contained about eight ounces of thin pus, "without the least remnant of the lungs." But the editor of the journal adds a footnote: "In all probability the lung was only very much compressed and closely bound down to the mediastinum so as to escape observation, a case which has occurred to ourselves.—Editor."

Beginning now, certain topics concerning pneumothorax began to occupy the attention of the thesis-writers, and one of the first is this one interesting group of the cases mentioned by Laennec. Laennec gave no good evidence that emphysema could cause pneumothorax. Devilliers undertakes to fill this gap.

50. Devilliers. Du pneumothorax déterminé par la rupture de la plèvre et d'une vésicule aérienne emphysémateuse. Paris, 1826.

After reviewing the history of the subject the writer tries his hand at classifying the forms of the disease, and then considers the one in question.

Case 1.—Emphysema of Both Lungs, Passage of Air under the Pleura, Rupture of the Pleura, Left-sided Pneumothorax.—The patient had a sudden attack of pain at night, followed by a sense of constriction and dyspnœa and in a few hours death. At the autopsy air could be

heard rushing out on opening the left chest. The lungs were very emphysematous, and on the left side the pleura had become dissected off at points, forming emphysematous blebs and one of these had ruptured. The author thinks death resulted from the inability of the other emphysematous lung to do the work of both.

Case 2 is Meckel's (q. v.), which he considers such a case as the above, but that the long compression had removed all traces of the emphysema of this the only affected lung. (But this case was more likely chronic fibrous tuberculosis. The pleura was somewhat thickened, there were some adhesions present, the lung was firm, dry, airless, and filled with hard masses.)

Case 3, (quoted from Cruvielhier, is more likely tuberculosis, although the emphysema, quite general and air bubbles in the blood vessels, would suggest the post-mortem result of a gas bacillus infection. See original article given in abstract.)

The symptoms in this group of cases are due: First, to the emphysema. Two sets of emphysema are, however, to be separated, that following rupture of the lung (interstitial), and that characterized by dilatation of air vesicles, the sort to which this paper refers. Second, the symptoms due to the rupture of the air vesicle and the pleura, of which sudden orthopnœa is the chief.

As complications of such cases he names, among others, pleuritic effusions and adhesions of the lungs.

This writer by reporting his case gives us perhaps the first case recorded of pneumothorax following pure emphysema. But in his choice of two other cases and in his remarks on them he shows that he included in this group cases not belonging there, for it is unlikely that one lung only would be the seat of this emphysema, or that compressing the lung would remove all traces of this disease. Rupture of an emphysematous bleb would neither cause an effusion nor the formation of pleural adhesions, as he supposes.

At this point it is interesting to read the thesis of

51. Warme. Les Plaies de Poitrine. Paris, 1826. (Hence he was a fellow-student of Devilliers.) This is a surgical topic and he quotes Petit and Boyer concerning the symptoms of perforation, but spends most of the time discussing the physiology of respiration, giving a very full and satisfactory résumé of the ideas of that time. But he does not mention pneumothorax nor even the names of those clinicians whose works he may have read, showing that the surgical condition of air in the pleura was not considered related to this new medical disease.

52. Andrew Duncan, Jun. Contributions to Morbid Anatomy. *Edinb. Med. and Surg. Jour.*, Vol. 28, page 302, 1827.

Ten cases of pneumothorax are reported—

Case I.—Simple empyema of the right side. The amount of pus was enormous and the lung seemed absent.

Case II.—Empyema of the left side.

Case III.—Empyema of the left side. This was evidently a case of pyopneumothorax. The woman felt something give way in her chest, the fluctuation in it was audible to bystanders. Mental symptoms were also present. At autopsy, however, no pneumothorax was found. The obliterated fistula was found. The writer describes the sounds heard during life, which he considers were metallic tinkling, (although he evidently did not know of Laennec's findings then.) He quotes Nysten, whose experiments proved "that large quantities of air are readily absorbed by the healthy pleura."

Case IV.—Simple pneumothorax of the right side. This was a terminal case of tuberculous pneumothorax without effusion.

Cases V-X.—Empyema with pneumothorax. In Case X the presence of H_2S was proven in the gas.

53. **Garde.** Observations de Pneumothorax. *Arch. gen. de med.*, 1828, XVII, p. 345.

This article is commonly referred to Rayer as author, in whose clinic the cases occurred.

Case 1.—A girl 23 yrs. old, suffering from phthisis. Onset of pneumothorax was six weeks before admission (these cases are most minutely reported), and from that time the only position she could assume with comfort was to crouch supported by her elbows and knees. She kept this position about fifty-six days, or until five days before death. Rayer's diagnosis was "Phthisis pulmonalis, opening of cavity into left pleura, communication of bronchus with this cavity, heart situated in right side of the chest." This dislocation of the heart was so great that Rayer considered it as a congenital abnormality, but autopsy showed it a dislocation due to the left pneumothorax. The cardiac pulsation was below and outside the right breast. The left chest was distended, gave a drum-like tone on percussion, remarkable amphoric respiration heard, "like blowing into a bottle."

The last six days she lay on her back, but the respiration was even more embarrassed than before.

At autopsy air rushed out from the chest. At first glance no lung was there, but it was found compressed against the spine and covered with false membrane. The fistula was easily found, open, the size of a large lentil, through which air could be easily blown. The heart was displaced to the right so that its apex rested against the sternum.

Case 2 was a woman 24 yrs. old, who had a sudden onset of pneumothorax, in the course of a chronic tuberculosis. She could lie but on the left side. The left side gave all the signs of pneumothorax, including amphoric respiration, metallic tinkling, and a splash on succussion which could be heard at some distance. The interesting feature was the extreme dislocation of the heart to the right. The patient recovered enough to walk about, and then died with sudden respiratory distress.

The perforation of the left lung was open, but found only after careful search.

The interest in these two cases for us is the fact that the dislocation of the heart was noted. The writer states that Laennec had seen it but once, that Louis does not mention it, and that John Davy saw it in one case. But we may perhaps be correct in ascribing to this writer the credit of having first called that attention to it which added it to the signs of left-sided pneumothorax.

54. Piorry. De la Percussion médiate, etc., 1828.

It is to be expected that Piorry should have tried his invention, the plessimeter, on every case at his disposal. Unfortunately, there was no case of pneumothorax at hand, so his observations were made on the cadaver. He allowed air to enter the chest and found the note became tympanitic, but of a quality which allowed it to be distinguished from that of the abdomen. If air be forced into the pleura the tympany is even more marked. He considers the following signs pathognomonic of air in the pleura, i. e. a very clear tympanitic tone, more marked if considerable fluid be present. One must exclude a distended stomach by the signs at the apex. He says Laennec doubted the value of percussion in making a diagnosis because he used only direct percussion. Mediate percussion, giving better differentiation of tone, is sufficient (on the cadaver) and he says Tarral confirmed his claim (also for a cadaver, but of a pneumothorax case).

Under the title, "Diagnosis of Pneumothorax," p. 91, he says one can sometimes hear respiration in a case of pneumothorax, which Prof. Cayol also found. "This is not surprising, since elastic fluids are conductors of sound, and the respiration murmur of the opposite side can be transmitted through the gas of the diseased pleura as well as through the tube of the stethoscope." Hence mediate percussion is more important in the diagnosis of this disease than is auscultation. He thinks on the cadaver mediate percussion allows one to find upper level of dulness of fluid in hydropneumothorax. Piorry recognized movable dulness, emphasizes it as we might expect, for it is by no means one of the lesser advantages of mediate percussion. He also considers that the presence of adherent parts of the lung may be thus recognized.

55. Williams (Charles J. B.). A Rational Exposition of the Physical Signs of Diseases of the Lungs and Pleura. 1828.

This little book contains an excellent discussion of the explanation of many of the phenomena of physical diagnosis.

P. 132. He divides cases of pneumothorax into two groups, the *active* and the *passive*. A pneumothorax is active when the air, whatever its source,

presses the lung down; it is passive when the air either entering by a communication with the external air supplies the place of the lung diminished by its own collapse, or generated within the sac fills up the cavity of a reabsorbed collection of liquid after those cases of pleurisy in which the lung is bound down by a fibro-cartilaginous membrane and the parietes of the chest cannot by their collapse obliterate the cavity. "This division includes and defines all the varieties of pneumothorax." Active pneumothorax is a rare occurrence and generally like symptomatic hydrothorax is the precursor of death.

In speaking of *metallic tinkling* he first describes the requirements of a good resonance chamber, and then says: "Pneumothorax may combine these conditions . . . the cavity is always present . . . but it is best calculated to produce the tinkling echo when its parietes are tense and regular, as when the lung is bound down by a fibrocartilaginous membrane. The sonorous impulse may be produced within the cavity or communicated to it from without. The only cause of the sound within the cavity would be of a portion of liquid contained within it dropping, on a change of posture or in the motion of respiration, from the upper to the lower parts. Such a case of metallic tinkling is rare, but Laennec records an instance and I have myself met with one." "Sounds external to the cavity may cause the tinkling echo within it. They may be communicated in several ways. Thus a fistulous communication with the bronchi may transmit a sonific impulse to the cavity and occasionally the speaking or coughing, and this is by far the most common cause of tinnitus metallicum. It is here most perfectly produced when the fistulous communication is short but narrow, and the bronchus is of considerable size. The sound is then heard immediately after the cough or utterance . . . sometimes but more rarely the same tinkling accompanies respiration." Amphoric resonance is obviously caused by the passage of air in and out of the pleural cavity. "This passage of air deranges the reverberation that produces the tinkling echo within the cavity, hence amphoric resonance either impairs or completely destroys the tinnitus metallicum." "Laennec considered the metallic tinkling pathognomic . . . (of open fistula fluid and air) . . . The accuracy of this opinion I am led by theory and experience to call in question. . . . The cavity being present . . . a cause of the tinkling echo in it may be found in the transmission of a sound or sonific impulse through solid parietes. For example, the voice or cough may communicate such impulse whenever by an induration or condensation of the pulmonary tissue it is rendered capable of conducting to the cavity the sound from any large bronchial tubes. Such a condensation . . . may be produced by inflammation and by the pressure of a liquid effusion, and might doubtless in some cases result from the pressure of the air of the pneumothorax itself . . . So far in theory.

"Now in the way of fact, Laennec himself furnished two examples which analytically prove the point at which we have arrived by theory. In one the tinkling was produced . . . where there was no communication with the bronchi or with the external air, by the dropping of the

liquid in a cavity. Another case presented the tinkling echo after the voice, although there was no communication with the bronchi" . . . (the wound was external).

Williams then reports a case of a boy, 15 yrs. old, evidently ill with pneumonia of the left lung, with right-sided pneumothorax in which after cough or utterance a distinct metallic tinkling was heard. He died (evidently from the pneumonia). "The lung was bound down to the whole of the posterior and upper portions of the lateral and anterior parietes of the thorax by a pretty firm fibrocartilaginous membrane which also thickened the costal pleura of the cavity. There was not a drop of liquid in the cavity, and there was no communication with the bronchi. In the inferior lobes of the compressed lung were found three hydatids contained in, but not connected with, a cavity lined with a fibrous membrane. The tissue of the lung was flaccid and compressed in their vicinity and bounding the cavity containing air, but above it was healthy and crepitant. The lower lobes of the left lung were found in a hepatized state which passed superiorly into simple inflammatory engorgement. . . . There were no tubercles in either lung."

"The rationale of the symptoms is in this case obvious; the tinkling echo was produced in the cavity by the voice transmitted to it by the pulmonary tissue condensed by the hydatids and perhaps by a former liquid effusion in the pleura. This effusion had been absorbed, and the lung being bound down by a factitious membrane, a *pneumothorax of necessity* was left."

He adds: "If I have shown that the stethoscopic signs are less certain than they have been represented to be, this may prevent future error."

Earlier in the book (page 123) he defines a "*pneumothorax of necessity*" as "A partial cavity . . . formed by an effusion which on its reabsorption cannot be filled by any contraction of the thoracic parietes (and by the lung since it is confined by a fibrocartilaginous membrane). It therefore becomes filled with gases with which the animal fluids abound."

56. Johnson (James.) Case of Pneumothorax, with an Account of an Operation Performed for its Relief, the Effects of the Operation, and the Appearance on Dissection. *The London Medical Gazette*, Vol. III, Jan. 31, 1829.

The writer was called to see a case "of scrofulous character," who was found lying on the right side. The dyspnœa was violent, respiration chiefly diaphragmatic. The left side was not larger "but sounded louder than natural and the right side sounded considerably duller than natural," etc. The next day metallic tinkling was the most important feature, "not only when the patient coughed or spoke but even during every inspiration and expiration." Nine days later five physicians consulted to consider the advisability of an operation for the "dreadful dyspnœa." "The action of the respiratory muscles was painful to behold and it was evident that but a very small portion of air could be taken in at each inspiration." In a footnote the writer adds, "Some of the medical gentlemen present, and particularly Mr. Johnson, compared the metallic tinkling to the

sound emitted by a musical snuff box, and this in reality is a more exact similitude than that which Laennec has employed." But the patient was so near death that operation seemed hazardous and much to his disappointment none was performed. But at once a consultation of other physicians was held, and "An incision was made in the anterior lateral part of the left side of the chest between the sixth and seventh ribs . . . a rush of air issued forth with a loud humming noise, strong enough to extinguish several candles had they been near the orifice . . . the relief was almost instantaneous." . . . No fluid escaped. The next day the patient was again in distress and the wound was opened by a director; air rushed out. Before this the metallic tinkling was perfectly audible, but "On examination of the left side immediately after the escape of the air no metallic tinkling could be heard by any of the gentlemen." The next day a canula was pushed into the wound and the finger put over its orifice during inspiration and removed during expiration when a rush of air always escaped. "This was continued until no doubt could remain as to the fact that part of the air drawn in by the mouth was thrown out of the wound at each expiration." He died the next day, and an autopsy performed. "Previous to the examination . . . a surgeon demanded of Mr. Johnson what were the morbid appearances which he expected to find? Although it was a question which it would not be always very charitable to ask before a dissection, yet Mr. Johnson did not decline the answer . . . The disease was pronounced to be "pneumathorax;" and the morbid appearances would be a collection of air and some other fluid in the left side of the chest; collapse of the corresponding lung; and aperture in the lung capable of giving free vent to air from the lung to the cavity of the pleura; displacement of the heart; probably tubercles of the right lung." The lung was found compressed to about one-fifth its normal size; fourteen ounces of turbid serous fluid was found. The fistula found by inflating the lung before removal was capable of admitting a crow's quill. The pathologist "formally declared that every iota of the diagnosis was verified by the dissection."

57. **Maréchal, A.** Observations de Pneumothorax reconnu par le bourdonnement amphorique et sans communication de l'épanchement aériforme avec l'air extérieur. *Jour. hebdom. de méd.*, Paris, II, p. 116, 1829.

Case 1.—Was a girl, 10 years old, suffering from typhoid fever. On the 10th day after admission signs and symptoms of partial right-sided pneumothorax developed, recognized by the amphoric resonance and sonorous percussion note. Metallic tinkling or succussion splash are not mentioned. At autopsy air rushed out from the right chest (opened by plunging in a bistoury) with considerable noise. It could be felt by the hand as it issued.

(This case is of interest since the description of the intestine leaves little doubt that it was typhoid fever.)

Case 2.—Also a girl, 10 yrs. of age, with very similar physical signs

of a pneumothorax developing twenty-five days before death. Here also at autopsy the hand could feel the gas issue from the opening made in the chest. No air could be forced through the lung, which was completely carnified, containing no air.

The writer sums up the cases as follows: Both were cases of pneumothorax manifested by the amphoric breathing, resonance on percussion, absence of respiratory murmur. At autopsy no tuberculosis, no gangrene, and no lesion in the pleura (tear) was found. He thinks therefore that Laennec is wrong who ascribes this amphoric resonance (on auscultation) to an open fistula.

But at the hands of Proust these cases have suffered severe treatment—he pronounces them pleurisy with effusion, and all the clinical symptoms are best thus explained. But to explain the air issuing from the thorax after plunging in a knife, this is harder to explain. Proust suggests post mortem putrefaction of the fluid or that it entered when the chest was opened.

58. Reynaud. Pneumothorax. *Jour hebdom. de méd.*, Vol. VII, 1830, page 81.

The author first gives a literature of practically all the recent articles on the subject. He proposes to consider its occurrence, whether more common on the right or left side. He collected all the cases in literature to date, forty-eight, and found it on the left side in 32, on the right in 17 (one was bilateral).

59. Hamilton Roe. Case of Pneumothorax in which Paracentesis was Performed, with an Abstract of a Clinical Lecture. *London Med. Gazette*, 1832-3, Vol. XI, p. 398.

The patient was a tuberculous case of four months' standing. Pneumothorax developed on the left side. At autopsy two or three perforations were found, one as large as a half crown.

60. Houghton. Account of a Remarkable Case of Pneumothorax. *Dublin Jour. of the Medical and Chemical Sciences*, 1832, I, p. 313.

The author doubtless gave the prevalent idea of the prognosis of pneumothorax when he said, "There are some lesions of so grave a character that peculiarity of constitution is esteemed to avail nothing against them; they progress with greater or less rapidity to the death of the patient, which is the only relief for the anguish that usually attends them without remission till it arrives." Perforation of the intestine and pneumothorax with a fistula connecting pleura and bronchus are examples of such lesions "which are analogous in their nature and resemble(ing) one another in generally being rather fatal in their results."

The man was a laborer, tuberculous, who had a suddenly developing pneumothorax of the left side with all the symptoms and signs common to that condition. He was very ill, and surprised his doctor by slowly

improving until he went to work with the pneumothorax still present. The left side had then contracted somewhat, the heart still displaced, and the succussion splash could be heard as he went down stairs.

"This interesting case furnishes the most surprising instance of resistance to extensive organic destruction and profound morbid action that I am aware of, and I believe that none similar is on record." He does not record this as a case of recovery, but as the first on record of such improvement. "When we consider these accounts (the experience of others), thi(s) individual . . . , with one lung compressed by air and fluid and its function destroyed, the other the seat of tuberculous deposition, and his heart displaced, presents a curious object for contemplation as he goes about his daily work as cheerily as those about him."

61. Berthe. PNEUMOTHORAX. *Thesis*, Strasbourg, 1833.

This thesis is a brief historical review of the subject.

62. Houghton. THE SEQUEL OF A CASE. . . . *Dublin J. Med. and Chem. Sc.*, 1833, III, p. 200.

The patient continued work as a brick-layer, for weeks, but grew worse, was admitted to the hospital July 21, left in October and worked more at brick-laying, but died January 25 (1833?). At autopsy the left chest (the affected) was 1¼ in. smaller than the right, the mediastinum was thickened. Three perforations were found. He is of the opinion that an attempt at cure was demonstrable.

63. Fuchs. PNEUMOTHORAX BEI TUBERKULOSEN LUNGEN. *Heidelberger Klin. Annalen*, 1833, p. 86, Vol. IX.

One case(a man 30 yrs. old) is described with autopsy.

This is one of the earliest German references.

64. Forbes. TRANSLATION OF LAENNEC'S DE L'AUSCULTATION MÉDIATE. 1834.

In the footnotes this editor adds much that is new and interesting, bringing the subject well up to that date.

Metallic Tinkling. Footnote page 56. "In one of the most distinct examples of this phenomenon that I ever met with no communication could be discovered on dissection between the bronchi and the fluids in the sac of the pleura."

As regards the formation of gas from the decomposition of fluid Forbes is decidedly skeptical for he says (p. 457) this idea of Itard and Laennec is decidedly problematical, that no doubt in Itard's cases the air entered through a fistula although it was not found.

To Laennec's causes of pneumothorax Forbes adds, the rupture from the pleura into the lung as in empyema and cites Archer's and Hawthorne's cases, but adds that an empyema is often thus discharged without causing pneumothorax.

To Laennec's list of physical signs he adds those described since Laennec's publication, the decubitus of the patient, the majority lying on the

affected side, the displacement of the heart best seen in left-sided pneumothorax, and the displacement of the liver in right-sided pneumothorax.

65. Houghton. PNEUMOTHORAX. *The Cyclopædia of Practical Med.* (Tweedie), 1834, Vol. III, p. 451.

This article was pronounced by Forbes, "a very valuable treatise."

The writer writing thus early in the development of the subject ascribes all the credit to Itard and Laennec. He defends the name pneumothorax on etymological grounds. It is of interest that he says: "It has never been satisfactorily proved and it seems to us an extremely problematical circumstance, that pneumothorax ever occurs by the putrefactive decomposition of a pleuritic effusion . . . It may be laid down as proved by the recorded experience of medical men that where pneumothorax exists the air has been introduced from without, for cases of an opposite description are so rare that they may be considered as exceptions to the rule."

Pneumothorax produced by the bursting of a tubercular abscess into the pleura. "If we were to conclude from the experience of the medical men of Dublin who have given most attention to the subject it might be asserted that (this cause) constitutes fully nine-tenths of the cases of pneumothorax, with the exception of the traumatic variety.

PATHOLOGY.—The writer remarks the infrequence of perforation of the summit where the cavities are most common and largest, and discusses the function of the elasticity of the lung in producing its collapse (adopting Carson's views), admitting that the case is not clear. Perforation is most common at the inferior part of the upper lobe or the superior of the middle, in the most cases on a line with the third rib but may happen at any part of the pulmonary substance.

The inevitable result of the perforation is acute pleurisy even though "scarcely anything but atmospheric air" enters the cavity.

The orifice of the perforation tends to increase in size and often attains the size of the tuberculous cavity that originated it, but it rarely passes the size of a shilling. The perforation is formed by the remains of a small cavity and the latter is often connected with other larger cavities by a bronchus, which opening obliquely converts the orifice into a valvular structure. He describes and explains the mechanism of these oblique fistulæ.

He says decomposition of the fluid never occurs, since if it did in a few it would in all. The odor he explains by the gangrene of the pleura.

As to the gas, he cites Apjohn's analysis of CO_2 8, O 10, N 82 per cent.

In the great majority of cases the orifice acts as a valve and the results are well described.

HISTORY AND DIAGNOSIS.—a. *Dyspnœa and Pain.*—He differs from Louis as regards the value of these as a certain diagnosis.

b. *Cough and expectoration.*

c. *Decubitis.*

d. *Dilatation of the side*, chiefly depending on the valvular nature of the orifice.

e. *Displacement of the heart and depression of the diaphragm.* "It appears that less compression is requisite to effect these conditions than to produce dilatation of the side. The perforation is more common on the left side, hence the position of the heart is very important. The low position of the liver is important and the sulcus at the costal margin is very valuable in distinguishing a depressed from an enlarged liver."

f. *Fever.* Constant.

Physical Signs.—a. *Succussion.*—The cause of this was not earlier recognized because "the early notions were a mass of confusion on this subject, but for the most part it was conceived that a vacuum existed in the thorax. This absurd hypothesis precluded the necessity for the presence of air to cause the fluctuation." . . . Morgagni believed that when no fluctuation is heard the space is "filled up."

b. *Percussion and Auscultation.*—Laennec is much quoted, but the writer emphasizes the value of movable dulness. He cannot translate the words "bourdonnement amphorique," hence uses them. He cannot agree with Guthrie as regards metallic tinkling.

Prognosis and Treatment.—He admits the possibility but considers the proof of cure of tuberculous pneumothorax still wanting.

Pneumothorax from gaseous secretion of the pleura.—He records the existence of this form merely on the authority of Laennec, considering it still unproven.

From opening of an empyema into the lung.

From gangrenous perforation and the rupture of the pleura in emphysema.

From lesions of the thoracic parietes.

1. Fractured rib, the "emphysema thoracis" of former writers.
2. From the rupture of an abscess through the intercostal spaces.
3. From the empyema operation.

66. Graves. An Effusion of Air within the Chest in Inflammation of the Lungs. *Dublin Jour. of Med. and Chem. Sc.*, 1834, Vol. VI, p. 49.

The case reported was that of a man 40 yrs. old, who caught cold and was attacked by cough, pain in the right side, blood expectoration, in short the usual symptoms of very intense pneumonia commencing in the lower right lobe and later involving the whole lung. "The chest was on the third day dull below and resonant above. On the next morning the chest was still dull below, but from just below the right nipple to the clavicle the percussion note which the day before was naturally sonorous, now yielded a preternaturally clear and hollow sound. No respiratory murmur whatever could be heard in this region and consequently we were led to the conclusion that the subjacent lung was here pushed back and compressed by air effused into the cavity of the pleura . . . At our next visit in about sixteen hours after, we found this area now as dull as possible and presenting a very obscure respiratory murmur mixed with some crepitus . . . We now felt no doubt that the air so suddenly effused had been as suddenly absorbed, and its place occupied by the inflamed and

engorged lung." The dulness steadily diminished and the patient recovered.

This case Proust condemns as pneumonia.

67. Graves. On the Accumulation of Air in the Cavity of the Pleura and in the Peritoneal Cavity, and on Emphysema after Profuse Hemorrhage. *Dublin Jour. of Med. and Chem. Sciences*, Jan. 1, 1834, Vol. IV, p. 309.

Frank, in de Curandis Hominum Morbis, treated the subject of the morbid development of air in the human body at great length and with his usual ability, but few seem to have studied this class of diseases with the attention which it deserves; and yet the improvement lately made in our knowledge of the laws which regulate the diffusion of gases and their transition or passage through the texture and membranes of the living body, and our more intimate acquaintance with the phenomena of secretion, ought to enable us to enlarge the limits of this important department of medical science.

He then argues that the body is not "air tight," but its whole substance is pervious to air and that a considerable portion of air constantly exists in the body during life. The names of Dalton in connection with pneumatosis and of the Americans, Mitchell and Graham, on the diffusion of gases, are mentioned.

Graves takes issue with Houghton regarding this gaseous secretion as a cause of pneumothorax, especially on Frank's authority. And cites two cases as proof of his position, arguing that in both the air was secreted.

68. Poole. A Case of Pneumothorax from Perforation. *Dublin Jour. of Med. and Chem. Sc.*, 1834, Vol. V, p. 185.

This was a case of tuberculous pneumothorax (left side, man, age ?), but what interested the writer were the signs of reparation on the part of the body. He takes firm stand against the secretion theory.

69. Martin Solon. Examination of the Gas contained in the Pleural Cavity of a Case of Pneumohydrothorax. *Arch. gén. de méd.*, Series II, Vol. 9, p. 464, 1835.

The patient was clearly pneumothorax following tuberculosis. At the autopsy the gas was analyzed qualitatively and a large percentage of CO_2 found, but quantitative analysis was not made. The writer considers a gas of such composition must be harmful to the body and advised its removal by puncture.

70. Stokes. Diseases of the Chest (1837), *New Sydenham Society*, 1882.

P. 559. After a general introduction he says, speaking of Louis' emphasis on *symptoms*, that one cannot trust these since he had frequently suspected pneumothorax but found the physical signs wanting. Speaking of *onset*, he says in one patient of his the first sensation was that of a sudden crack extending from above downward and accompanied by a feeling as

if liquid were shed out into the chest; acute pain in the side afterwards set in.

Again he says: "In several instances I have observed a complete change in the character of the cough and a cessation of expectoration. The latter symptom seems peculiar to those cases in which the expectoration had been furnished by the lung, which was subsequently perforated. I have seen a case in which the expectoration, being previously copious, ceased on the occurrence of the fistula and only returned when tubercular ulceration had invaded the opposite lung." One case suffering often from dyspnœa due to increase of the liquid effusion, "relieved himself by the extraordinary manœuvre of placing his head on the ground and then elevating his heels against a wall until the reversed position was nearly vertical, when a vast quantity of seropurulent fluid was expectorated and relief given for a considerable period of time."

Two cases of pure pneumothorax are mentioned to show that air alone causes no inflammation.

He emphasizes the valvular nature of some cases and says Duncan first showed that it is the oblique course of the fistula that explains the valvular action. But such cases are rare (the pressure increasing until death) and the majority of cases in less than a week exhibit a freedom from suffering which is very remarkable. Such cases he considers as open pneumothorax.

He thinks a valvular fistula may become an open one; he rather doubts that the reverse can occur.

Concerning the effusion he makes two classes. In the one, the more common, the liquid remains stationary for months; in the other it increases until it causes distress. In one such case the patient was able to relieve himself by inverting his position and allowing the fluid to run out of his mouth.

He believes that once open a fistula is always open, and the evidence for its closure in some cases is unsatisfactory, and he believes that closure would help little, since the patient will die of the disease in any case.

Stokes now speaks of the remarkable cases in which the onset of pneumothorax is followed by the suspension of the constitutional symptoms of phthisis. He mentions one good case in which the improvement was extraordinary, and the man was even better than before his illness, and said "if it were not for the annoyance of a splashing sound in his chest, which attracted the attention of his partner in the waltz and of his companions during equestrian exercise, he suffered no annoyance whatsoever," yet all the signs of pneumothorax were present, while he was confident of his restoration to health. "Of the temporary suspension of the symptoms of phthisis there are now many examples," and in case the other lung is not involved it "really produces a temporary cure of consumption," but he considers it only temporary, although the man may live for months or even a year. Finally the man will sink; it may be that the fluid then begins to increase, to a dangerous degree, or the opposite lung

becomes involved, or, as in one case described, the "lymph lining the pleura itself may become transformed into tubercular matter. The cessation of phthisical symptoms is best marked when the collapse or compression of the lung has been most complete, and this is more the case in early cases than when the lung is extensively diseased." He mentions a very rare case in which the perforation was at the very apex of the lung; usually this part is adherent.

Stokes also believes perforation of the lung and of the intestine are in several ways analogous; the onset in each is sudden or latent. The pain and distress at the onset of pneumothorax cannot, he says, be due to the exclusion of the lung from respiration since it is not a feature in occlusion of a bronchus; it may be the nervous excitement, the spasm of the opposite lung, or the sudden pleurisy.

Of the effect of air on the pleura Stokes makes the significant remark, "We know that the entrance of air by the canula during paracentesis is a matter of trivial moment."

The diminution in expectoration after the development of a pneumothorax, "which contributes to the fallacious appearance of recovery from consumption" is not wholly due to the passage of the sputum into the pleural cavity, but its diminished formation in the collapsed lung. But it may even be felt by the patient to fall into the pleural cavity after coughing.

On the other hand, the discovery of a pneumothorax in a phthisical patient is often a pure accident.

Regarding *physical signs*, he says that the more normal the lung the greater will be the change in the lung signs.

The *metallic sound* may be given to all acoustic phenomena, voice, heart, respiration, râles, etc., or to one, or a few. He is quite sure one of his cases showed metallic tinkling due to the dropping of fluid within the cavity; again in other cases this tinkling may be synchronous with respiration and impress one as due to the bursting of bubbles in a fluid.

The percussion tone varies much: near the middle of the chest it usually has an exaggerated tympanitic resonance; above it is modified by the lung, below by the fluid. Those cases in which with much fluid present the chest is hyperresonant to the base he attributes to the paralysis of the diaphragm which then forms a pouch containing the fluid. The *succussion splash* may be easy or hard to obtain. He has seen cases in which it could not be produced, while the patient sat up in bed but could easily if on a chair. In cases with very much fluid we have tympanitic dulness. Stokes then quoted the first case of pneumothorax recognized in Dublin since the discovery of mediate auscultation, in which case metallic tinkling was audible to the patient but could be heard by the examiner only through a stethoscope. Dr. Graves suggested the sounds of a musical snuff box as the best likeness for the sounds, but Stokes thought a tuning key better. The fistula opened under the surface of a large effusion. The fistula would admit the little finger and was two inches from the apex of the lung; there was another large one at the base, and possibly three others.

The description of six cases then follows:

Case I.—Chronic phthisis, sudden perforation, with consequent empyema and pneumothorax continuing for five months. Notwithstanding the chronicity the fluid did not become truly purulent.

Case II.—Phthisis, with consecutive fistula, empyema and pneumothorax; operation for empyema, subsequent gangrene of the pleura.

Case III.—Acute phthisis with pneumothorax. He died three weeks from onset.

Case IV.—Simple empyema from injury, perforation of the lung and consequent empyema and pneumothorax.

Case V.—Gangrene of the lung, empyema, and pneumothorax, paracentesis; gangrenous destruction of the costal pleura; passage of the fluid behind the peritoneum. In this case the gangrene had laid bare the intercostal muscles at the posterior inferior part of the right chest and the fluid made its way beneath the skin down the abdomen even to the scrotum.

Case VI.—Empyema of the right side, opening by anthrax; pneumothorax by external fistula. This case is particularly interesting. The empyema perforated under the right short ribs. Pneumothorax followed. The cough, voice, and the breath sounds caused by the air entering the fistula were metallic, yet every drop of fluid had drained out. Hence he agrees with Williams that for their production a cavity alone is necessary.

Stokes discusses the relation between whooping-cough and pneumothorax, but considers tuberculosis always present.

Stokes is pessimistic of the value of operation, considering it a last resort and liable to lead to gangrene of the pleura.

71. Lemaire. Pneumo-thorax. Paris, 1837.

(This brief thesis contains a general review of the subject, but also the report of one case which is almost unique, since it was a case of pneumothorax resulting from abscess of the liver.)

A young man, 18 yrs. old, admitted in the service of Andral, who previously had been treated for a suppurative process in the testicle, was admitted in a condition of shock. No diagnosis was made. Two days later he had a severe pain in the base of the right chest, followed by dyspnœa. The right side was tympanitic on percussion, even to the summit; marked amphoric respiration, some metallic tinkling. He died three days later. The autopsy showed the right chest almost full of pus with a little gas anteriorly. The pus was yellowish green, with flecks of fibrin. The lung was collapsed and could not be inflated, but gas could be blown through a hole in base of the lung. The liver was adherent to the diaphragm. In the liver were several abscesses, one of which had perforated into the pleural cavity.

There were no tubercles in either lung.

72. Tanquerey. Pneumothorax. Paris, 1837.

This thesis is merely a review of the subject.

73. Pneumothorax from Fracture of the Clavicle. *Med. Examiner*, August, 1838.

A man, 46 yrs. old, fell from a roof, fracturing the clavicle. Right-sided pneumothorax resulted; recovery.

74. **Mercier.** Perforation of the Right Lung, probably due to a Hydatid. Hydatids in the Right Pleural Cavity and Liver. *Bull. de la Soc. anat.*, Vol. 13, page 71, 1838.

The case is that of a man, 27 yrs. of age, admitted for hæmoptysis. The clinical history is well given. Three weeks after admission the patient had a sudden attack of pain in the right side, and the following day the diagnosis of perforation of the lung was made from the physical signs.

At autopsy (death two weeks after onset) the pleural cavity contained air and serous fluid in which was a hydatid cyst. The lung was perforated. In the liver was a hydatid cyst. There was no tuberculosis of the lung.

75. **Thompson (A. T.).** Case of Pneumothorax. *Lancet*, Apr. 20, 1839.

The case was that of a woman, 29 yrs. old, whose history indicates tuberculosis, although the writer says, "There was every reason to conclude that fluid and also air were contained in the serous sac, but as there were no obvious symptoms which indicated the existence of phthisis I could not decidedly pronounce the case to be one of pneumothorax." The diagnosis, (made it would seem chiefly on the absence of the respiratory murmur and metallic tinkling), was confirmed at autopsy. The air in the pneumothorax was "found to contain three times more CO_2 and one-half less O, than common air." The perforation was open. "The right lung contained not more than ten or twelve tubercles scarcely softened; and the perforation evidently had arisen from the ulceration of one of them, which lay close to the surface of the lung." He considers that the false membrane resulting from an old pleurisy had prevented the formation of the adhesions which usually protect the lung in such cases, and he thinks this a case of very early phthisis and that therein lies the chief interest of the case. There were no symptoms to date the time of perforation.

76. **Bigelow (Jacob).** Remarks on Pneumothorax, with Cases, and an Experimental Inquiry into the Causes of the Metallic Sounds heard in that Disease. *American J. of the Med. Sc.*, Vol. XXIII, p. 45, 1839.

"Metallic" phenomena is a very good name for sounds heard after the disease his existed for a considerable time. They are heard during respiration and cough, also with the voice, succussion splash, and on percussion. The sound is sharp and tinkling or reverberatory and ringing according to the action causing it, but the mechanical conditions producing it are the same.

The sounds are (1) those of impulse—in which case fluid must be present; (2) those of reverberation for which only air need be present. The first includes all tinkling heard in respiration, after speaking and coughing, and can in some cases be produced abundantly by succussion. When well developed it is sharp, musical, silvery, like the snapping of short, brass wires in certain children's toys. The second includes amphoric breathing, the metallic resonance of the voice and cough and of the percussion tone.

For the metallic tinkling, one of the most marked physical signs, all the explanations are unsatisfactory.

1. Laennec's explanation of drops of fluid falling, not accepted now.

2. Dr. C. J. B. Williams said they were due to the reverberation or echo of sounds in a cavity. If the fistula be small there is produced only metallic tinkling; if large, amphoric resonance.

3. Davies (*London Med. Gaz.*, Vol. XV) ascribed them to the resonance of air on the surface of a liquid. This is similar to the explanation given by M. Meriadec Laennec, who said the gas vibrated on the surface of the liquid.

4. Dr. Jas. Houghton said it was the echo both from the dropping of fluid in the cavity and from the bursting of bubbles of air entangled in mucus at the orifice of the fistula.

5. Guthrie (*London Med. and Surg. Jour.*, 1833) said that to the above factors must be added great compression of the air.

6. Beau said it was caused by bubbles of air rising through the fluid and bursting at the surface. and is never heard when the fistula opens above the surface. (The same idea expressed by Spittal in 1830.)

7. Magendie (*Lancet*, 1835) disproved all these theories by experiments on the cadaver, but suggested no new one. He was unable to reproduce these sounds on the cadaver, putting in the chest a certain amount of water and then blowing air through the lung, etc.

Bigelow says he knows Guthrie is mistaken, since the tinkling may be present in a large tuberculous cavity with fistula, and that Davies is too vague. The "echo" idea is unsatisfactory, water being a better conductor of sound than air.

Bigelow now reports three cases of pneumothorax and then proceeds to describe his experiments on the bodies at autopsy:

Exp. II.—Artificial respiration was maintained through the trachea. At each inflation the same distinct metallic sounds heard ante mortem were produced. This was not true in Case III, but ante mortem in this case the sounds were only faint.

Exp. III.—A catheter was inserted into the chest and air blown in. If the orifice was above the surface of the fluid only amphoric buzzing was heard, but if below, exquisite metallic tinkling. These were more easily produced with little than much fluid.

Exp. IV.—Percussion and succussion produced the same sound on the dead as on the living body. The metallic sound heard on percussion somewhat resembled those occasionally yielded by the heart (Bouillard).

Exp. V.—None of these phenomena could be produced on a body that was not before death a case of pneumothorax (see Magendie's work).

Exp. VI.—A bladder and a stomach were filled with water and fluid. Then a tube was inserted and air blown in. If the end was below the surface of the water a sharp tinkling was heard which varied in intensity with the tension of the bladder. If the end was above, an amphoric sound was heard. If saliva be blown in, a feeble submetallic tinkling was heard.

Conclusions.—For the metallic tinkling to be produced:

1. "There must be a cavity the walls of which are preternaturally susceptible of vibration. This takes place when the pleura is pathologically distended so as to overcome the obtuse or muffling effect of the contiguous soft organs, such as the lung, diaphragm, and the intercostal muscles. Some time is probably necessary to prepare the parts for this pathological resonance, since it fails to appear post mortem in healthy chests submitted to experiments. It should be added that when metallic sounds appear in simple phthisis, there are cavities of the lungs the walls of which are the seat of tuberculous induration."

2. The immediate cause is the forcible disturbance of liquid in such a cavity, especially by air from beneath the surface, but also liquid thrown up in the act of coughing, which falls back. This is also the case in succussion.

3. The vibrations are transmitted by the liquid to the walls without the necessary agency of echo or reverberation.

4. Submetallic tinkling having a musical resonance is produced by slight impulses given to the air in the cavity, e. g. by the breaking of mucus bubbles.

5. Amphoric resonance is caused by the reverberation of air in a vibrating cavity, without the sonific impulse of air. This is also true of the metallic modification of the voice and of cough.

Metallic percussion also seems to depend on the vibration of the air independent of the liquid and may be produced in some other cases when we strike upon a tense cavity in which a certain quantity of air is confined.

77. Williams (C. J. B.). Pneumothorax. *Dissertations on Diseases of the Organs of Respiration.* Tweedie, 1841.

Pneumothorax may be produced in three different ways:

1. It may be the consequence of a partial pleurisy. In an old case of pleurisy "the absorption of liquid leaves a void which the collapse or contraction of the walls of the chest is in some cases insufficient to obliterate and this void is sometimes filled by air secreted by the membranes. We have seen two instances of partial pneumothorax produced in this way . . . This kind of pneumothorax is very rare."

2. "Another kind of pneumothorax is that which may be called idiopathic, and arises from an effusion or secretion of air into the sac of the pleura without perforation. This is also of very rare occurrence. It is said to occur sometimes towards the termination of fatal diseases in the same manner as tympanites occasionally occupies the peritoneal sac under

similar circumstances. We have never met with such a case in which the signs of pneumothorax were observed during life, but we have several times seen a little air in the pleural sac when it is opened after death without any discoverable perforation of the pleura . . . Pneumothorax is also said by Drs. Hudson, Graves, and others to have occurred in a few instances at the commencement of pneumonia, and to have afterwards disappeared; but as the chief sign in such cases was a remarkable resonance on percussion we suspect that these were examples of the production of tracheal or amphoric sound from consolidation of the upper lobe of the lung, and not cases of pneumothorax."

3. Perforation of the pleura. Under physical signs he says: "We have heard the metallic tinkling accompany both the voice and the cough in a case of partial pneumothorax without liquid effusion or perforation of the pleura, the sound being transmitted to the cavity through the condensed tissue of the lung. But it is where the pleura is perforated and where liquid is present that the phenomenon of metallic tinkling is commonly heard; not as Laennec supposed because these conditions are essential to its production, but because the motion of the liquid or of the air through the orifice make sounds within the cavity which serve to show its echoing properties."

"In listening for the metallic tinkling it must be held in mind that they may be audible only in certain parts of the chest where the lung is not adherent and where the liquid effusion does not reach. Generally in the sitting posture they are best heard about the mammæ and the lower part of the scapula and axilla; but we have heard them in some cases in every part of the affected side and in others only in one spot. In fact there must be a certain degree of tension in the walls of the cavity to make them good reflecting surfaces, and if this be deficient at the spot of the cavity opposite to that on which the stethoscope is applied the sound may be absorbed and not reflected."

Williams, however, believed that "by percussion we can find the exact level to which the liquid rises." "On change of posture and on coughing the liquid will sometimes drop from the parts which have just been immersed; and the sound of this will exhibit the metallic ringing in so distinct a manner that it resembles the note which a glass or porcelain vessel yields when struck."

Concerning the splash on succussion, he says, "the splashing is not easily produced unless there be a good deal of air in the pleural cavity with a moderate quantity of liquid."

Gérard in 1865, pronounced the following thesis the most complete till that date, including as it did all the cases on record at the time of its publication.

78. **Saussier.** Researches on Pneumothorax and the Diseases which produce it, especially Perforations of the Lung. *Thesis*, Paris, 1841.

(After the appearance of Laennec's book, cases of pneumothorax were reported from all sides. In 1830 Reynaud collected forty-nine, but eleven years later this author was able to collect in all literature, old and recent, one hundred and sixty-nine.)

As this review of Saussier was considered a very important one, we will quote at length.

Pathological Anatomy.—The cases he divides into:

1. Traumatic. Those from

a. Puncture of the lung by a fractured rib, or by any instrument penetrating the chest and lung. Three cases are cited, those of Littré, Hunter, and Méry.

b. Rupture of the lung by contusion of the lung without lesion of the chest wall. The cases of this rare form are Hewson's and Smith's but in the latter case the details given are too meager to allow of just criticism. (*Dublin Jour.*, 1840.)

c. Rupture of the pulmonary vesicles from severe pressure on the chest. He gives one case.

d. Penetrating wounds of the chest wall without lesion of the lung; as in the operation for empyema.

2. Non-traumatic.

a. Phthisis pulmonalis. Eighty-one cases collected.

b. Pleurisy. Twenty-nine cases collected.

1. With effusion, the gas formed by the pleura.
2. With effusion, the gas formed from the decomposing fluid.
3. Without effusion, the gas exhaled by the pleura.
4. With effusion, the fluid perforating through the lung, and the air entering through this perforation.

c. An effusion of decomposing blood. One case is cited.

d. Pulmonary gangrene:

1. The gas entering through the perforation formed by the gangrene.
2. The gas formed by the decomposition of the gangrenous tissue.

e. Pulmonary apoplexy, causing perforation. One case is cited.

f. Rupture of emphysematous vesicles:

1. The superficial vesicles together with the pleura rupturing. Five cases are cited.
2. The vesicle but not the pleura ruptures, hence there arises an "extrapleural pneumothorax." One case.

g. Pneumonia terminating in abscess, rupturing the pleura. One case.

h. Hydatids of the lung opening both pleura and bronchi. Three cases.

i. Hydatids of the liver opening both pleura and lung. One case.

j. Ulcerating cancer of the lung. One case.

k. Ulcerating cancer of the stomach which perforates the diaphragm and lung. One case.

l. Abscess of bronchial lymph glands which perforates the lung and bronchus. One case.

m. Simple perforation of the bronchus, not due to perforation of the lung or bronchial glands. No cases.

n. Cyst of the pleural cavity. One case.

o. Rupture of the œsophagus. One case.

p. Essential pneumothorax, the development of gas in the pleura not due to any of preceding lesions. One case.

q. Pneumothorax from post-mortem putrefaction. No cases.

r. Disease not indicated. Four cases.

Pneumothorax with Pulmonary Phthisis.—In 81 of 147 cases it was on the right side in 25, left in 50 and bilateral in 1 case; not stated in 4. Of these the presence of the perforation was noted in 72 cases; the situation in 47 cases; the upper lobe in 28; middle in 7, and lower in 12. It was stated to be closed in only 6 cases. The maximum length of fistula was 5 cm., the largest were 2-3 cm. in diameter. Of 61 cases in which the number of perforations were noted it was single in 47, there were 2 to 6 in 13, and in one case the lung was fairly riddled.

Pleurisy.—Of 37 cases of pleurisy with pneumothorax and perforation of the lung due to pleurisy without pneumothorax, pneumothorax was found in 29. Of these 29 the perforation was found in 13, was absent in 8, doubtful in 2, not stated in 6, hence in 8-10 cases the gas existed without discoverable perforation.

Of the 21 cases of pleurisy in which the lung was perforated, in 17 cases it was on the right side, in one on the left, in 3 not stated (contrast with tuberculous cases).

Combalusier's and Meckel's cases he considers as examples of cases in which the gas came from the pleura, for "it is pretty nearly certain that there was neither perforation of the lung nor phthisis present." The author states that many deny the existence of this form, but does not think that one is justified in doing so. He cites five cases in which "it is necessary to resort to the exhalation of gas to explain the foundation of the pneumothorax with which they are accompanied." These cases were:

1. Pouteau, *Œuvres posthumes*, t. 1, p. 341, Obs. 6.
2. Vénot, *Thesis*, 1824.
3. Maréchal, *Jour. hebdom.*, Vol. 2, p. 116.
4. Maréchal, *Jour. hebdom.*, Vol. 2, p. 219.
5. Author's case. A patient 24 yrs. old with acute pleurisy on the left side and pneumo-hydropneumothorax on the right. (Hippocratic succussion was present.) The autopsy confirmed the diagnosis, but no perforation could be found by examination or on insufflation.

Pneumothorax from Decomposing Blood.—He cites Littré's case. He thinks it more probable that in this case the gas entered through the wound, but admits the possibility of such cases.

Gangrene of the Lung.—It is surprising that pneumothorax does not result from all such cases with perforation of the lung. Of 8 such cases there was no pneumothorax in 3. It is usually a terminal affair. In tuberculosis the lung tissue is destroyed and the pleura ruptured, but in this disease both lung and pleura are destroyed. The slough usually produces a large superficial wound opening many bronchi, but may produce a deeper loss of tissue with one free bronchus. The gas may also be

furnished by the decomposition of the gangrenous tissue, in which case it will be fetid.

Pulmonary Apoplexy.—There are only three cases on record, Bayle's, Corvisart's and Andral's. He does not think it an important variety.

Emphysema.—One of the most curious but best proven varieties. The first cases reported are Cruveilhier's and Devillier's. The sub-pleural form differs from the true only in extent.

Abscess of the Lung.—Only one sure case on record, Dalmas'. An abscess is usually expectorated through the bronchi.

Hydatids of the Lung and Pleura.—Three cases on record, and one in doubt.

Fouquier, *Clinique des Hôp.*, t. 2, p. 69. This was the case of a woman 36 yrs. old, with right-sided pneumothorax of sudden onset, confirmed at autopsy.

Mercier's case.

Fréteau, *Jour. gén. de méd.*, Vol. 43, p. 124. A case of hydatid of the pleura opening into the lung. The man, 28 yrs. old, was operated on for empyema. In the next few days over 500 hydatids escaped by the wound and by mouth.

Hydatids of the Liver.—Saussier knows of but one example and that very clear. He reports the case of a woman 42 yrs. old, with a long history of hepatic trouble. Right-sided pneumothorax developed suddenly. At autopsy a hydatid cyst of the liver had broken into the pleura and through the lung.

Abscess of the Liver.—Andral's case, 1836 (reported in the thesis of Lemaire, 1837).

Ulcerating Cancer of the Lung.—Andral in his lectures stated several of such cases were on record.

Cancer of the Stomach.—The air may come from the stomach alone or also from the lung. Andral's case is cited.

Tuberculous Abscess of the Bronchial Lymph Glands.—Such usually open into the bronchi, but may also into the pleura. Berton said it was possible, but gave no cases. Rilliet and Barthez (*Arch gén. de méd.*, 1840), give a case, but here the pleura was opened through the lung. The reason given for the infrequence of such cases in children is the tough fibrous sheath around tuberculous glands and cavities which directs the perforation into the bronchi.

Perforation of a Primary Bronchus into the Pleura without lesion of the lung or lymph glands.

Saussier thinks there are three possibilities of the perforation: (1) from a foreign body; (2) from an external cause; (3) from an ulcer (tuberculous or luetic) of the bronchus. No good illustrations of any of these forms is given.

Pleural Cyst.—The case given by Vénot (*Thesis*, 1824). (Proust pronounces it pneumothorax.)

Rupture of the Œsophagus.—Boerhaave's case is the only one on record.

ESSENTIAL PNEUMOTHORAX.—"We are inclined to admit this variety of pneumothorax (without apparent lesion), but have not the evidence to establish it beyond question."

POST-MORTEM PUTREFACTION.—He wishes "only to state the unquestioned influence of putrefaction on the development of pneumothorax and to call attention to the gaseous exudation without apparent lesion which is recognized only on the cadaver."

THE GAS IN PNEUMOTHORAX.—1. Quantity. Davy obtained the most (220 cu. inches). The most is found in those cases with a perforation 2-3 mm. wide and with a valve formed of pseudo-membrane.

The amount depends:

(1) On the condition of the lung. If normal it can collapse to the size of a small egg, but if hepatized or tuberculous it cannot.

(2) On the amount of fluid. These vary reciprocally; if the fluid increase the gas will decrease.

(3) On the dilatability of the chest. The greatest dilatation is seen in the valvular cases.

2. Composition. Analyses are given in eight cases:

Davy, 2 cases.

Bierre de Boismont, 2 cases.

Louis.

Portal, who introduced air during the empyema operation. At operation an inflammable gas with the odor of H_2S was found.

Martin-Solon.

Thomson.

THE EFFECT OF GAS.—1. On the lung. This will collapse or be compressed according to its elasticity.

2. On the pleura there is almost no effect. The sudden pain and dyspnœa are due to the rupture of the pleura. He quotes Davy's conclusions.

FLUID EXUDATE may or may not be present. Of 147 cases in all but 16 it was present.

STATE OF THE LUNGS.—These collapse and then are compressed. Often they cannot be reinflated. There are three grades of compression. In (1) the vesicular respiration is audible; in (2) the respiration is bronchial; in (3) there no respiratory murmur.

In 75 cases the position of the collapsed lung was stated: In 4 it was anterior, 12 superior, 75 against the vertebral column, and 5 floating.

The volume of the lung varies. It may not be compressed at all, even only partly collapsed and collapse still more when the chest is opened. It may be the volume of a hen's egg, and so small and covered with false membrane that it is reported absent.

SYMPTOMS.—The invasion was sudden with violent pain and dyspnœa in 68 of 196 cases; in 4 the sensation of rupture of the lung was present.

PHYSICAL SIGNS.—These are enumerated. Under *percussion* he says that the hyperresonance usually found may not be present but the affected side be duller than normal in case pneumothorax supervene on a

case of pleurisy with thickened pleura, even though the amount of fluid be small. The tone will be duller over the collapsed lung. Movable dulness is evidence of fluid.

Saussier describes *claquement costo-hépatique*, a sign which bears his name, as positive proof of pneumothorax, especially on the right side. This is the sensation of the eighth rib when percussed striking the liver. It occurs only when there is much gas and the liver is not depressed. It is due to the vacant space between the rib and liver, left by the retraction of the lung's margin. It can be present on the left side. He thinks in some cases it may be of service.

Among the *auscultatory signs* are those of respiration, cough and voice, metallic tinkling, amphoric resonance, and the succussion splash. For Laennec's confusing classification he proposes:

1. Vesiculo-amphoric respiration, which is the vesicular murmur slightly modified by a small amount of air in the pleura.

2. Broncho-amphoric respiration, when the lung is compressed.

3. Fistulous-amphoric respiration, when air can freely enter and leave the pleura through the perforation.

4. Amphoric crepitation, or metallic tinkling, when the air enters the pleura, passing through the liquid exudate in bubbles which burst at the surface.

5. The cough and voice are modified similar to the respiration. These sounds may be amphoric without being metallic, and considerable confusion exists in the use of these terms. Laennec's theory of metallic tinkling is given up and Dance's now held. It can be reproduced by blowing air into the chest through the trachea, the bubbles rising to the surface of the exudate, where they burst.

Duration.—Stated in 91 cases. 3 died in a few hours, 38 in less than 6 days, 20 less than one month, 19 in from 1 to 5 months, 2 in from 8 to 11 months, 1 in 18 months, and Barlow's case lived 3 years.

Prognosis.—Pneumothorax is always a serious complication, and he argues at length against the existing opinion that pneumothorax should have a favorable influence over the tuberculous process.

Treatment.—For the pain, morphia and blisters; for the congestion, venesection; for the dyspnœa, he advises operation to allow the air to escape. In seventeen cases this was followed by satisfactory results. To Combalusier, 1747, and Riolan he gives the credit of advising operation.

79. Rokitansky. Pathologische Anatomie, Vol. 3, 1842.

Page 55. Rokitansky in this chapter cites no cases. He gives a list of causes of pneumothorax, but does not state how many cases of each he has seen. As his book purports to be a record of his experience, we assume that among his 12,000 autopsies he saw pneumothorax resulting from:

1. Rupture of a tuberculous abscess.

2. Gangrene.

3. Rupture of a subpleural tubercle.
4. Pulmonary abscess.
5. Decomposition of an exudate.
6. In rare instances is found a certain amount of air with a benign pleural exudate, the result of pleurisy.
7. The erosion of a bronchus.
8. Rupture of emphysematous vesicles; or the tearing of the pleura in a case of interlobular and subpleural emphysema.
9. The perforation of the diaphragm or of the mediastinum from acute softening of the stomach or œsophagus.

80. Chomel. Pneumothorax. *Dict. d. Med.*, Vol. XXV, 1842.
This is a good review of the subject, but contains no new point.

81. Skoda. Percussion and Auscultation. 2nd Ed., 1842.
Page 292. Skoda considers that air can cause an exudative pleuritis.
Signs on Percussion.—"The percussion note in pneumothorax is usually distinctly tympanitic. If, however, the chest wall is under considerable tension, the percussion note is little or not at all tympanitic. In most cases is also heard the metallic tinkling. It is often not loud enough to be heard at a distance, so one must combine auscultation with percussion. The fluid present at the same time in the pleural cavity with the air or gas always occupies the lowest part of the chest and changes its position with every change of the patient. But the fluid must be present in considerable quantity in pneumothorax to be recognized by percussion. The percussion note is still tympanitic, even below the level of the fluid, and is not at all changed by even a rather thick layer of fluid. To estimate the amount of fluid present in pneumothorax it is therefore necessary to double the amount indicated by percussion.

Foot-note.—Apart from this case, combined percussion and auscultation, that is the auscultation of the percussion tone, has no value.

"Auscultatory Signs—On auscultation in pneumothorax one hears either the amphoric respiration, or the metallic tinkling of the voice or respiration . . . ; or one hears no one amphoric resonance and metallic ring, but an indefinite respiratory murmur (with loud, dry and moist râles), a dull echo of the voice; or finally one hears none of the voice, no respiratory murmur, etc. These differences in auscultatory signs do not depend on the fact that in the one case the air in the pleura communicates with bronchi, in others on the other hand is perfectly closed off. I have never seen a case of recent pneumothorax in which the communication between the pleura and bronchi was preserved. I found it always closed, partly by the compression of the lung, partly by the exudate. Only after the pneumothorax has existed some time can, in very rare cases, a communication between the external air and that in the pleura arise by ulceration. The difference in the auscultatory signs in pneumothorax . . . depends on the fact that the air-containing pleural cavity is sepa-

rated from the bronchus sometimes by a thinner, sometimes by a thicker layer of lining parenchyma, and in it the voice, the respiratory murmur, etc., 'resonate.'"

Page 130. Amphoric Respiration and Metallic Tinkling.—"These arise in the thorax only when there is present a large air chamber whose walls are suited to reflect the tones... (hence they occur) only when large cavities or pneumothorax is present."

"Laennec imagined that the cavity must contain both air and fluid for these signs to be produced, and to my knowledge nobody has yet denied this. I believe that the fluid is unnecessary."

Laennec believed also that for these signs to be produced a communication with the bronchus was necessary. But "in pneumothorax it is only in the rarest cases that the communication between the air in the pleura and bronchus remains free, and yet it is seldom that these signs are not present." They also occur in cavities in the lung parenchyma, but he has never found them in a cavity smaller than an average sized man's first.

In pneumothorax the metallic tinkling is commonly caused by the intensification by the resonance chamber of a râle in a neighboring bronchus, or by the shaking of fluid, or rarest, the falling of drops into the fluid; and the amphoric respiration by the intensification of tracheal respiration.

Page 283. Speaking of the percussion tone above a pleural exudate, Skoda says: "The lung gives, when collapsed or compressed but still containing some air, a more tympanitic—sometimes a clearly tympanitic—and sometimes a louder tone, than the normal inflated lung."

(This is the Skodaic resonance which has led many to diagnose pneumothorax when no air was present.)

82. **Fiebiger.** De pneumothorace, adnexis morborum historiis synopticis. Prag, 1842.

In this thesis (Latin) are reported three cases of tuberculous pneumothorax with autopsy notes.

83. **Ehrenburg.** Ueber Pneumothorax. Würzburg, 1842. A thesis with the report of a case of tuberculous pneumothorax.

84. **Saussier.** Nouvel exemple de pneumo-thorax traumatique, et réflexions sur cette maladie. *L'Expérience*, April 28, 1842, Vol. IX, p. 257.

The patient, a man 32 years old, was run over by a wagon, fracturing eight of the ribs of the right side. Pneumothorax resulted and subcutaneous emphysema. The breath sounds were absent, there was no metallic tinkling. No fluid effusion followed. The patient was able to leave the hospital quite improved. He thinks the pneumothorax was a favorable feature, since it kept the chest immobile.

He found here his sign claquement costo-hépatique valuable in diagnosis.

85. Leas. Pneumothorax in a Child two years of age. Fatty Liver and Tubercular Deposition. *Dublin Jour. of Med. Science*, 1843, Vol. XXIII, p. 167.

This tuberculous child screamed considerably one night and died the following morning. At the autopsy considerable air gushed from the left side of the chest. The heart was found pushed to the right of the midline. At the angle of the fourth rib was the perforation into a tuberculous cavity. Immediately above the opening was an adhesion. Both lungs were studded with tubercles. The liver was a typical "fatty liver." This was the youngest pneumothorax case on record at that date.

86. Bruchausen. De Pneumothorace. Gryphiae, 1844.

This thesis reviews the general subject and gives seventy-eight literary references, many of which are not often given. He reports a case of simple pneumothorax in a case of phthisis, with recovery.

87. Dechend. Pneumothoracis monographia, addita una de hoc morbo observatione. Halae, 1844.

An excellent review (in Latin) with abundant literary references, and the report of one case of the tuberculous variety.

88. Jadelot. Pneumo-thorax chez un sujet affecté de phthisie pulmonaire. Mort. Quelques considérations sur cette maladie dans l'enfance. *Gazette des Hôp.*, Paris, 1844, Vol. VI, p. 469.

The case described was a girl 9½ years old, who died of tuberculosis with pneumothorax. The perforation was found; he thinks it due to the rupture of a superficial tubercle. He discusses the disease, considers obliteration of the perforation rare. There is nothing new in the discussion of the case.

89. Wach. De pneumothorace. *Thesis*, Prag, 1846.

A good review of the subject, but containing no new cases or new work.

90. Marais. Recherches sur les Bruits anomaux du pneumothorax. *Thesis*, Paris, 1847.

In this thesis is the experimental work reviewed to date (Gérard).

"Since Laennec we must confess that no one had said anything which makes easier the recognition of pneumothorax. He was one of those persons who go to the bottom of the subject with which they deal and who leave little to be done in the region through which they have passed. But if he said the last word concerning the existence of the diagnostic signs of the disease he did not do so complete work in the interpretation of some of them."

For the *etiology* of this disease Saussier's thesis is quoted, adding little except Grapin's case due to gangrene.

Under pleurisy as a cause he says that it was not generally admitted,

even was vigorously contested, that pleurisy could be the cause of pneumothorax either by the exhalation of gas or changes in the exudate. He proceeds to deny that the cases given as illustrations were idiopathic, but thinks that in such cases the fistula heals, allowing the lung to expand as the exudate is absorbed, hence at autopsy no perforation is found. "Saussier seems to us to ascribe to the pleura too important a function in the production of these gases, although he points out the possibility of this phenomenon as a result of the transformation of fluid. He admits the presence of a mixed effusion in some cases, but ascribes the formation of gas exclusively to the pleura and gives as reason his assertion that two or three ounces of . . . fluid are incapable of producing enough gas to fill the pleural cavity." "It is rational to admit the united action of the pleura and fluid, but to deny the possibility of the production of enough gas to fill the pleura by two or three ounces of fluid . . . is to ignore one of the best established and recognized laws of physics," considering the enormous volume which liquids take in the gaseous transformation.

Further on he says that at the moment air enters the pleural cavity it has been warmed somewhat, hence expanded; in the chest its tension will be even more increased, but after death this gas will contract (since cooled), hence that gas rushing out after death cannot be that which was present during life, but must be produced post mortem.

Symptoms and Physical Signs.—(This section is of especial interest, for, since the writer has so much to say about physical laws, we presume he was well up in the physics which will explain the physical signs.)

The expansion of the gas distends the chest, causing its immobility. The percussion note may not be changed or duller than normal, but in general it is hyperresonant and drum-like. Percussion also determines the amount of fluid, the extent of pleural adhesions and the position of the compressed lung. Laennec was so pleased with mediate auscultation, his discovery, that he neglected percussion, which gives more information than he admitted, and is an important auxiliary of auscultation, especially as it is the sign which usually first calls one's attention to the condition.

Auscultation.—(The most important part of the paper.) Without agreeing with those who with Laennec think an open fistula necessary for amphoric respiration, he decidedly disagrees with Saussier that the sounds of a partially collapsed lung which is lax is capable of transmitting well characterized vibrations. He would explain Saussier's findings by the presence of adhesions holding some of the lung in contact with the chest wall and that here the vesicular respiration arises. He thinks Saussier was misled by preconceived notions. He denies that the fistula has any rôle in producing sounds.

Metallic Tinkling.—The opinions of all preceding writers are discussed, Laennec's dropping theory, Dance's and Beau's, that the fistula must open under the surface of the fluid; Fournet's that the narrowness of the fistula was important, and Castelnau's. Granting the possibility

of the above by way of argument, Marais shows their conclusions impossible. Suppose that air bubbling into the pleural cavity through a fistula with submerged orifice did produce metallic tinkling during inspiration, it could not during expiration, for the discrepancy in size between the pleural cavity and the bronchial tree is such that for air to bubble from the former to the latter would flood the bronchial tree with the pleural exudate before the level in the pleura was low enough for air to leave it, and supposing it could bubble into the bronchial tree, that being a resonance cavity of very different properties from the pleura would not produce metallic tinkling. He cannot agree with Laennec that metallic tinkling and amphoric resonance have the same cause. He doubts that fluid could fall in drops from the summit of the chest into the underlying fluid, for it would, even when tags of membrane are there, plaster these to the chest wall and flow along the side without dropping.

Raciborski said metallic tinkling was due to the collision of particles of the fluid, agitated by respiration and hence considered fluid and an open fistula necessary. But Marais says it does occur, in cases without fluid. He gives a case to illustrate this latter point.

(Marais is very unjust to Bigelow, stating that his idea was the same as Raciborski's and that he confused various sounds, and not mentioning Bigelow's experiments or his conclusion concerning the necessity of an abnormal pleural cavity.)

Castelnau concluded that metallic tinkling is only mucous or cavernous râles resounding in a spacious cavity and is evidence in favor of an open fistula, and exudate may or may not be present. This explains its occurrence with each phase of respiration, its occurrence after unusual efforts, e. g. cough, but does not explain those cases without a fistula or with one that is submerged.

Guérard explains it by the sudden entrance into the pleural cavity of a certain amount of air forcing before it at the mouth of the fistula mucus or detritus collected there. But this does not explain its presence where there is no fistula, nor the fact that the metallic tinkling occurs at the end of inspiration, while amphoric resonance occurs at the start, showing the fistula open.

Routier reviewed opinions as follows:

1. The metallic tinkling and the amphoric resonance cannot be produced by one bronchial fistula.

2. They occur in simple pneumothorax with perforation and in hydropneumothorax provided the fistula is above the surface of the fluid.

3. There is no true metallic tinkling if the fistula be below the surface of the fluid, but then one hears a simple "gargouillement argentin."

4. It is the momentum of the penetration of gas into the pleural cavity which produces amphoric resonance.

5. The metallic tinkling is due to the bursting at the fistula's orifice of a bubble of gas enclosed in mucus.

But Marais insists that both phenomena occur when the orifice of the fistula is above the fluid, below the fluid, or when there is no fistula

at all. Barth and Roger say each explanation may in certain cases be true. Marais suspects them all false.

Marais' explanation is based on the experiments of Castelnau and the opinion of Skoda.

If air rushes from the chest at autopsy, this indicates that free exit had not been allowed it. The experiments of Castelnau and of Marais showed that if the lung were punctured, air could be injected through this fistula, but could not be withdrawn, hence in the great majority of cases the alternate entrance and exit of the air into the pleural cavity does not exist, hence the fistula does not enter into the production of these sounds.

Marais adopts Skoda's idea that all that is necessary is a suitable resonance cavity.

The conclusions of the writer are these:

1. The hyperresonance, distension, and immobility of the thoracic walls are easily understood, since air usually cannot escape from the chest (hence is under tension).

2. The absence of the respiratory murmur is explained by the collapse of the pulmonary parenchyma.

3. For the *amphoric respiration* a fistula is necessary; it is enough that the walls of the cavity be capable of entering into vibration with the current of air set up, not necessarily by air entering the cavity, but even by the respiration of the sound lung. The intensification of amphoric resonance at the end of expiration is explained by the higher intrapleural tension which obtains in the pleura at this time, hence the chest is more resonant. He cites his animal experiments to prove this point. The intensity of the amphoric resonance depends on the amount of gas in the pleura, the distension of the thoracic walls, the degree of compression of the lung tissue, and on the respiratory power of the individual. The last element is illustrated by its disappearance before death.

4. Metallic Tinkling also is produced entirely in the chest and transmitted to the ear by the distended thorax. It is a râle produced in the broncho-tracheal tube which is related to the pleural cavity much the same as a tuning fork is to its resonance box. If with a stroke of the bow one sets the tuning fork into vibration, it gives a resonance which, although louder, is comparable to the amphoric respiration. If while giving the fundamental tone it is hit a quick, light blow with a metal rod there is introduced a new sharp sound, brief and silvery, which gives perfectly well the idea of metallic tinkling. The condition most favorable for the existence of metallic tinkling is the accumulation of a large amount of gas in the pleural cavity. It should fail nevertheless in the absence of the râles which produce it, and is present when the fistula is closed. A case to illustrate this is given.

5. The cough and voice sounds are silvery ("voix argentine"), are produced in the larynx and communicated by the bronchial tree and solid lung to the distended chest.

6. The succussion splash is best heard when there is relatively much air in the chest.

7. The frémissement métallique is produced by the bubbles of air enclosed in the fluid when the chest is violently shaken. This "strepitus argentin" is not at all comparable to the succussion splash.

DIAGNOSIS.—Sudden pain and dyspnœa may indicate pneumothorax. The expectoration of costal fragments is a sure sign of pulmonary perforation, as also is the expectoration of fluid injected into the pleural cavity. In large cavities rarely occur the amphoric respiration, metallic tinkling, or even hyperresonance, but if they do occur the chest is not so immobile and the air enters and leaves more freely.

91. Grisolle. TRAITÉ DE PATH. INTERNE, Vol. I, 3rd Ed., 1848, p. 792.

Grisolle includes pneumothorax among the pneumatoses, considering that there is no doubt concerning the existence of essential pneumothorax but that this gas is secreted only by an inflamed pleura. The prognosis is good.

92. Hotte. DIE PATHOLOGISCH-ANATOMISCHEN VERHAELTNISSE DES PNEUMOTHORAX NEBST ZWEI KRANKENGESCHICHTEN, Würzburg, 1848.

The classification of causes of pneumothorax is quoted from Rokitansky, and his and Mohr's names recur repeatedly in the following pages, which contain a discussion of the pathological anatomy of this disease. He reports, Case I. The cause was, he says, not tuberculosis, but either bronchiectasis or empyema. Case II was pneumothorax caused by fractured ribs (the man was run over by a wagon) with recovery.

93. Crawford. IDIOPATHIC PNEUMOTHORAX FOLLOWING DIFFUSE INFLAMMATION OF THE ARM. DEATH. *The British-American Journal of Medical and Phys. Sciences*, Jan., 1848.

The patient's arm became very painful and much swollen following a cut at the wrist. Fifteen days later right-sided pneumothorax suddenly developed. Seven days later he died. The autopsy showed right-sided pneumothorax, the lung was compressed and airless. There was no trace of fluid in the pleural cavity. There was no sign of disease of that lung on inflating it. The other lung was much congested.

The writer considers this a case of idiopathic pneumothorax. He will not suggest a cause but discusses the pneumatosis theory (secretion of gas).

94. Fromm. PNEUMOTHORAX. Berlin, 1849.

This thesis is simply a review of the subject.

95. Bender. DE PNEUMOTHORACE, 1849.

This thesis contains, besides a brief review of the subject, one case with right-sided pneumothorax due to gangrene of the lower lobe posteriorly.

96. Rumpf. BEITRAG ZUR LEHRE VOM PNEUMOTHORAX. Würzburg, 1849.

The author reports five cases and discusses the causes of the physical

signs observed on them. Four were tuberculous cases (and the first was perhaps, although he rejects that diagnosis), considering pneumonia, bronchiectasis with gangrene, the sequence.

He discards the theory that metallic tinkling is due to the dropping of fluid into the effusion. After an opening is made into the chest the lung takes part in respiration for a while, the entrance or exit of air in the pleural cavity depending on the size of the fistula. A small opening is easily closed by expiration, which results in a more rapid filling of the cavity with air, and a quicker compression of the lung . . . but at first with each inspiration the fistula is opened and the bubble of mucus or exudate which closes the orifice while it is collapsed bursts. This rupture of a bubble gives rise to the metallic tinkling. A similar sound is produced by bubbles of air rising from the orifice of a submerged fistula and bursting at the surface. The explanation of other physical signs present in his cases was attempted.

97. Milcent. La mode de production du tintement métallique, du souffle amphorique, etc...*Revue méd.-chirurg. de Paris*, Vol. V, p. 323, 1849.

The author proposes to strengthen the theory of Skoda, advancing new arguments in its favor.

Criticism of Theories. Laennec gave three causes: (1) open fistula in a case with liquid effusion, (2) falling of drops from top of chest into fluid, (3) external fistula. But the presence of liquid is not constant, also free communication between the pleura and bronchus does not exist. Castelnau's and Marais' work are now quoted as showing that the fistula takes no part. Other reasons are given, showing the insufficiency of the theory that it is a sign of the triple lesion.

The second and third causes assigned are as inadequate as the first.

Theories of Dance, Beau, Fournet, etc. These authors modify Laennec's first explanation. The open fistula is necessary but it must open under the surface of the exudate. Dance says, "A certain amount of air passes during the acts of speaking, coughing, breathing through the bronchopleural fistula and gushes to the surface of the pleural exudate, forming bubbles of various sizes which burst at the surface of the fluid, setting in vibration the elastic fluid contained in the pleura and giving to it the character of the resonance characteristic to metallic tinkling." Castelnau disproved this theory.

Beau explained them by the passage of air through the purulent material of the fistula which opens above the level of the fluid.

Fournet claimed that metallic tinkling was produced if the opening of the communication was very small and below the surface of the liquid, the amphoric resonance if large and above the level, and an intermediate sound when the orifice was of medium size, and if two fistulæ are present, a large above and a small below, the sounds coexist. Milcent reviews the fistula theory as follows:

1. The hypothesis of a free communication between the pleura and bronchi is insufficient, for there are cases where the metallic tinkling and amphoric resonance are heard when there is no perforation.

2. The hypothesis contradicts certain important facts, among which is the high tension of the intrapleural gas.

3. It also contradicts experimental evidence and the simple laws of physics, etc.

Vol. VI, 1849, page 65. The author proposes to replace all other theories by that of Skoda, which is quoted at length. The rest of the paper is occupied with a discussion of Skoda's, Marais', et alt., views.

98. Janssens. Observation d'un pneumo-thorax traumatique, suite d'une grande chute. *Ann. Soc. méd.-chir. de Bruges*, Vol. X, p. 157, 1849.

A man, 59 yrs. old, fell 50 feet. No serious immediate injuries were found, but very soon pain in the right side and dyspnœa were experienced, although nothing was found on examination. Five days later, however, he suddenly felt a severe pain in the right side with orthopnœa. He died of asphyxia that night.

At autopsy right-sided pneumothorax was found, the pleural cavity was about one-fourth filled with serous fluid, four ribs were found broken, but the pulmonary wound was not found.

99. Hérard. (Metallic Tinkling in the Kidney.) *Bull. de la Soc. anat.*, Vol. 25, p. 98, 1859.

A woman had a large tumor in the left side of the abdomen, resonant on percussion. On auscultation, if percussion were used at the same time, metallic tinkling could be heard.

At autopsy, in place of the left kidney was found a tumor 25 x 14 cm. in size filled with purulent fluid, and a gas containing equal parts of CO_2 and SO_2. The ureter was patent.

In the discussion **Barth** said that it was certain that bubbles of air rising to the surface of fluid could cause metallic tinkling, but it also occurs where there is no external communication.

100. Hérard. (Metallic Tinkling in a Large Tuberculous Cavity. *Bull. de la Soc. anat.*, Vol. 25, p. 322, 1850.

The cavity occupied almost the whole of the upper lobe of the lung and was larger than the fist. Its internal surface was very smooth, its shape almost spherical. It communicated with the bronchus by a hole about the size of a pen, and contained both fluid and air. During life were heard a loud amphoric murmur and metallic tinkling with in- and expiration. He called attention to the absence of the factors which Barth and Roger and Guérard considered necessary for their production, and believed these phenomena due to the coarse râles at the mouth of the fistula, and to the bronchial breathing, both reinforced by the smooth-walled resonance cavity.

In the discussion of this paper **Barth** hinted that cavernous respiration had been confused with amphoric resonance.

101. Kuentzelmann. De pneumothorace. Leipzig, 1850.

This thesis seemed to offer little new.

102. Ch. Bernard. Observation de pneumo-thorax. *Compt. rend. Soc. de biol.*, 1850. Paris, 1851, II, 1 pt., p. 189.

The lung was exhibited of a man who died twelve hours after the onset of the condition. He had had a tuberculosis of several years' duration. The perforation was in the superior part of the lower lobe of the left lung.

103. Delosea. De pneumothorace. Berlin, 1851.

In this thesis the writer discusses chiefly the question of essential pneumothorax. The English writers especially are quoted. He reports one case of "pneumothorax due to rupture of an emphysematous vesicle" in a man fifty years old. The man had had some cough and expectoration preceding. The onset was very sudden, the dyspnœa extreme and death followed in about three hours. At autopsy right-sided pneumothorax was found. (That lung was evidently tuberculous as well as emphysematous.)

104. Monneret. Sur le Pneumothorax et les phénomènes acoustiques auxquels il donne lieu. *Arch. gén. de méd.*, Mar., 1851.

The writer will add no new theory; his experience leads him to agree exactly with Laennec's ideas on these points.

Case 1.—Acute pneumonia of the left lung, terminating in gangrene and perforation of the lung; hydropneumothorax. The perforation of the lung came less than twenty-four hours before death. Intense amphoric respiration and metallic tinkling were present.

Case 2.—Tuberculous pleurisy followed by hydropneumothorax. In this case the amphoric respiration was very intense, although at autopsy the fistula was found filled by false membrane.

Case 3.—Phthisis pulmonalis, tuberculous excavation opening into the pleura; hydropneumothorax. The amphoric respiration and voice sounds were intense but there was no metallic tinkling. At autopsy four perforations were found, allowing free entrance of air into the pleura. There was scarcely any fluid in the pleural cavity.

Metallic Tinkling.—For the production of this are necessary (1) fluid capable of being set into vibration, (2) a suitable resonance chamber. The succussion splash is only a metallic tinkle heard at a distance. The sound is due to the sonorous vibration of particles of fluid. An entirely similar sound can arise in the stomach; those of cavities differ in quality, being less resonant. The bronchial, tracheal, et alt., râles are of the same origin and differ only in the size of the resonance chamber. The thoracic wall, he thinks, takes no part in their production. If it did one could dampen them by placing the hand against the chest, which is not the case. They (metallic tinkling) can be beautifully produced in vessels with stiff walls. The increase in these sounds during coughing, etc., is due to the partial closure of the glottis, hence air is forced into the pleura, setting the fluid into vibration. But no fistula is necessary, providing the fluid and air be in the same cavity.

Amphoric respiration is merely tubular respiration in a larger cavity

(or tubular respiration reinforced by a large cavity). The fistula need not be open. He does not agree with Skoda, but with Laennec, that these sounds are wholly similar to metallic tinkling; except that here it is the vibration of the vocal cords which is transmitted (Spittal). The collapsed lung is a conducting body, not a vibrating agent.

The amphoric voice and cough are only the sounds produced in the glottis transmitted through the bronchial tree to the pleural air.

The vocal fremitus he considered absent.

105. Burrows and Kirkes. *Med. Times and Gazette*, 1852.

The effect of pneumothorax depends on the previous condition of the lung whether in the full performance of its duty or not. He accepts the origin of gas from decomposition of fluid and by spontaneous generation independent of any previous disease of the pleura. Sometimes on opening the left chest air escapes which arises from the autodigestion of the stomach.

Case 1.—Tuberculous pneumothorax. In this case at the spine of the left scapula was amphoric respiration, and on deep inspiration a bubbling or popping sound, hence the fistula was here.

Four other tuberculous cases are reported at length.

106. M'Dowel. Adhesion of the Pericardium; Pneumothorax. *Dublin Q. Jour. of Med. Sc.*, Vol. XIV, p. 221, 1852.

The viscera of a case were exhibited. The boy (19 years old) had been in the hospital three months with left-sided pneumothorax with effusion. The onset was evidently gradual. The perforation was at the posterior surface of the lung at the juncture of the middle and lower thirds. The lungs were tuberculous. There was an old obliterative pericarditis. The heart was hypertrophied. The valves normal. The rare presence of cardiac hypertrophy in a case of tuberculosis with pericarditis is the point emphasized.

107. Hughes. Cases of Pneumothorax. *Guy's Hosp. Rep.*, 1852-3, 2 S., Vol. VIII, page 1.

Hughes first reviews a previous paper. He now reports—

Case 1.—*Caries of the Dorsal Spine—Abscess of the right Iliac region—Empyema—Pneumothorax of the left Pleura, communicating through the spine with the abscess—Pneumonia—Phthisis—Death—Inspection.*

He considers the caries of the spine the cause of the empyema and the psoas abscess. There was "a passage, in truth, existing between the mouth and the right iliac region, which passage . . . opened through the left lung."

Case 2.—*Tuberculous pneumothorax.*

Case 3.—*Pneumothorax, with pleural effusion of probably three months' duration.*

Case 4.—*Pneumothorax with pleural effusion—Sudden death after ten days—No inspection.*

Case 5.—*Pneumothorax with curved spine and Emphysema of the lungs, probably arising from rupture of distended air cells—No inspection.*

Case 6.—*Bronchitis and Emphysema—Rupture of the Pleura and Pneumothorax without fluid effusion—Paracentesis—Relief—Return of the complaint—Death—Inspection.*

"The edges of the lung were fringed with bullæ of emphysema of various sizes, from a pigeon's egg to a small pea." When paracentesis was performed a lighted candle held near the trochar was blown out. No evidence of pleurisy was found.

Case 7.—*Phthisis—Pneumothorax and Pleural effusion—Paracentesis twice performed—Cure of Pneumothorax—Suspension of Phthisis of that side—Death from Phthisis of the opposite lung—Inspection.* . . . The fact that the pneumothorax arising from a phthisically diseased lung was cured in consequence of the healing of the perforation, he considers makes this a unique case.

These cases, all but one of whom he had seen, are interesting because of the care with which they were studied and the unusual variety of interesting features they presented.

108. Margerie. (A New Method of Analyzing the Gas Contained in the Pleural Cavity in Pneumothorax.) *Le Moniteur des Hôpitaux*, Apr. 26, 1853, Vol. I, p. 393.

This communication is evidently from Trousseau's clinic. The apparatus pictured to collect the gas over mercury is simple. The gas of one case was tested, with paper moistened with subacetate of lead for H_2S or $(NH_4)_2S$; the CO_2, absorbed by a piece of KOH, was 35 per cent; O tested by a piece of phosphorus was absent.

109. Koechling. De pneumothorace. Bonn, 1854.

This thesis is a general review of the subject with brief mention of two cases of the tuberculous variety.

110. Banks. Pneumothorax with Effusion; Recovery. *Dublin Q. Jour. of the Med. Sc.*, 1854, XVII, p. 318.

The patient, a man 30 years old, was evidently tuberculous. In a paroxysm of cough he felt something in the chest give way and heard the noise. He could feel the heart move to the right and heard a splashing sound. The pain was agonizing, the dyspnœa extreme.

On succussion the splash could be heard across the room. The fluid and air both disappeared. It was about two years before the heart had returned to the normal position.

The author reviews the literature of this disease. For his case he claims it one of cured tuberculous pneumothorax and arrested pulmonary tuberculosis. The sound of the rupture had been heard in but three previous cases—those of Louis, Stokes and Thompson.

111. Wintrich. PNEUMOTHORAX. *Virchow, Pathologie und Therapie,* Vol. V, page 336, 1854.

Source of the gas.

Amount and nature of the gas.

Is the gas an inflammatory agent? As a result of a long series of experiments on various animals he decides that no gas except H_2S causes pleurisy. Usually it is absorbed in three days. After introducing any pure gas a mixture of O, N and CO_2 was always found, in varying proportions.

PHYSICAL SIGNS.

1. Pneumothorax with pressure symptoms.

(a) Without effusion—a rare form.

In such cases with the gas under tension the percussion tone is tympanitic. By simultaneous percussion and auscultation can the long axis of the cavity be determined.

(b) With effusion. With Skoda's rule that the amount of fluid present is twice that determined by percussion he cannot at all agree. Movable dulness is important. The change in pitch on percussion on opening the mouth allows the diagnosis of an open pneumothorax to be made. An increase in fluid may be determined from the change in pitch, etc.

(c) Hydropneumothorax as a result of pleurisy.

2. Pneumothorax without pressure symptoms.

3. Pneumothorax with retraction of the chest—always a pneumopyothorax, etc.

But under DIFFERENTIAL DIAGNOSIS the most interest lies. As a result of his numerous errors in diagnosis he decides that the recognition of pneumothorax is not so easy as is usually supposed.

To recognize extensive vesicular emphysema there should be no difficulty.

Extensive cavities are not so easy to exclude. He has heard the Hippocratic succussion splash for several weeks in a large cavity; also he cites a case of localized pneumothorax mistaken for a cavity. In such cases one should try movable dulness and the change of pitch on percussion, yet in one case with these signs tried he mistook a local pneumothorax for a cavity.

Subphrenic pneumothorax may cause confusion.

Bronchiectasis even to the base of the much contracted lung may confuse.

Pneumonia and pleurisy are sometimes mistaken for pneumothorax.

Pneumonia can be mistaken for local pneumothorax. In the course of a lobar pneumonia one hears in the center of a dull area, or over, under or beside it, a tone so clearly tympanitic, so pure, and metallic, that it is mistaken for pneumothorax. Such a tone he found always anterior, most often at the apex or just below the clavicle or in the mammary region. But the vocal fremitus persists, the râles are not metallic, the respiratory sounds are not metallic. At the apex the percussion note can sound beautifully metallic, Williams' tracheal tone, but the pitch changes when the mouth is opened and closed, which in pneumothorax never occurs.

Williams' tracheal tone may deceive in pleurisy but the change of pitch on opening the mouth, and the absence of movable dulness shows the condition.

All the other features of this disease, course, prognosis, etc., are discussed.

At the beginning of this article the author discusses the various physical signs in diagnosis of pulmonary diseases at length and reports his many experiments on them.

Wintrich emphasized the fact that the most frequent cause of pneumothorax was the rupture of a tuberculous excavation which breaks down before adhesions form, hence are not made out physically.

112. **Viedebantt.** De pneumothorace. Berlin, 1855.

This thesis contains no cases, no original work, almost no literature.

113. **Vallin** (in the service of **Malherbe**, to whom by some the article is ascribed). Observation de pneumothorax, consecutif à une pneumonie lobulaire terminée par abcès ouvert dans la plèvre. *Jour. de la sect. de méd. de la soc. acad. Loire-Inf.*, Nantes, 1855, XXXI, p. 98.

A woman, 42 yrs. old, was admitted to the hospital in a wretched condition but without apparent organic lesion. Signs of bilateral bronchitis, then right-sided pleurisy, then of pneumothorax developed. About eleven days after the last complication, the patient died. Autopsy was performed forty hours later. The gas was collected from the pleura and tested for H_2S and CO_2, both of which were present in abundance. The perforation was found opening from the pleura into a cavity about the size of a hen's egg, and in the consolidated part of the lung.

The cause of the cavity is discussed. The writer is confident it was not tuberculosis and decides it was abscess.

114. **M'Dowel.** On an Unusual Form of Pneumothorax. *Dublin Hosp. Gaz.*, 1856, n. s. III, p. 227.

The case was one of "*Sudden occurrence of pneumothorax on the left side, arising from the accidental rupture of an air vesicle; dry pleuritis; absence of tubercular disease; recovery.*"

A man, 33 years old, in quite good health, although with cough for two months, during a fit of laughing felt "a sudden burst in his left side." Immediately he was seized with intense pain and dyspnœa.

"The right side was remarkably and unusually tympanitic to percussion. This abnormal clearness of the stroke sound extended in front across the sternum, and replaced the normal dulness of the cardiac region. The heart had undergone an unusual displacement toward the right side. The left side was dilated and exceeded the right in circumference by at least half an inch; it also appeared smoother and was almost motionless during respiration. The respiratory murmur was almost extinct in the left lung, but no abnormal sound could be detected, neither metallic tinkling, splash on succussion, or amphoric respiration. The pleura therefore seemed distended with air without any admixture of fluid."

The writer considers the case non-tuberculous since in three years the lungs showed no signs of disease, and quotes Brünnicke's case as similar.

(This case is stated in full, since Proust says this was a case of pleural effusion and that M'Dowel was deceived by Skodaic resonance. We have heard of no cases with such general Skodaic resonance as this, and suspect Proust's obsession that there cannot be air in the chest without fluid, deceived him.)

115. Flint. PNEUMOTHORAX. *Buffalo Med. Jour.*, Vol. XII, 1857.

This was a case of spontaneous pneumothorax in a peddler with recovery. There were no signs of fluid, yet metallic tinkling was heard on the fifteenth day.

116. Skoda. PNEUMOTHORAX. *Allgem. Wiener med. Zeitung*, June 9, 1857, page 113.

The *etiology* is first given.

THE MECHANICS.—At first the lung collapses, air entering during inspiration and expiration until the lung is just collapsed. There will then be no distension of the chest, the liver will pull the diaphragm down somewhat. During inspiration air enters the pleura, which cannot escape during expiration, since the orifice is smaller or closes. Thus the chest is distended, and the mediastinum forced to the other side. The pressure is still further raised by the formation of an exudate and the warming of the air.

Partial pneumothorax is produced when there are adhesions in the pleural cavity. In partial cases occurs very often paralysis of the intercostal muscles, since the small amount of gas causes unequal tension of the soft parts. These paralyzed intercostals are drawn in on inspiration and forced out on expiration.

In the majority of cases a fluid exudate also forms. For a diagnosis it is necessary to determine:

1. The pushing down of the diaphragm and liver.

2. In a fresh pneumothorax, as a rule, the displacement of the mediastinum. If right-sided, the apex beat is displaced to the left; if left-sided, to the right and the stomach and spleen are displaced downwards.

3. Next to be determined is that the air present is not in the lung. This means the differential diagnosis between pneumothorax, emphysema, pleurisy and pneumonia.

June 16, p. 119. First must be determined that the cause of the symptoms is not a simple pleural exudate. If pneumothorax, the percussion note is clearly tympanitic unless the chest wall is under too high tension. If considerable fluid is also present, the presence of both may be detected by the movable dulness. But if but little fluid is present it will not be detected by percussion, and if the dulness is found the probable amount will be double that indicated by the line of dulness.

The succussion splash is a certain sign. The exclusion of emphysema is not so easy, the percussion tone is not decisive, but the amphoric echo

heard if the percussion tone be ausculted, is quite certain. The auscultatory signs are more conclusive yet not certain (they are reviewed). In pneumothorax one may hear a distant bronchial respiration, resonant râles with an amphoric echo, and metallic resonance of the voice, but no vesicular respiration, no near râles; but both the latter may fail.

In a fresh pneumothorax, since the air in the pleura does not communicate with that in the bronchi (the perforation being closed by compression), the various auscultatory murmurs depend on the thickness of the lung separating the pleura from the bronchus.

In pneumonia, tuberculosis, and bronchiectasis the bronchial respiration will distinguish them from pneumothorax.

June 23, p. 125. Course and Prognosis.—These depend much on the etiology. If the pneumothorax be due to rupture of the lung the contents of the pleura will be air and CO_2; O is well absorbed, CO_2 slowly, and N improbably, yet is found absorbed experimentally, as Skoda showed years ago on rabbits, into whose pleura he allowed air to enter; it was soon absorbed. This is true also of men. Healed cases of pneumothorax occur, as for instance the railroad employee treated in Prof. Schuh's clinic for rupture of the lung following his being crushed between two cars.

The danger of pneumothorax is not the air but the pleurisy which soon develops, but which may fail entirely in emphysematous cases.

In case of open fistula the amphoric respiration can be heard, or intense bronchial respiration. If the fistula contains also fluid the râles and metallic tinkling can be heard.

If the fistula opens below the level of the fluid, inspiration causes some air to bubble into the pleura, and expiration sends some fluid into the bronchus, which is promptly coughed up. This is repeated till much fluid is evacuated. During this process during inspiration often metallic tinkling may be heard, and after much fluid has been emptied amphoric respiration replaces it. Such cases have bad prognosis.

Therapy.—Skoda mentions a prevalent idea that it is well to advise external pneumothorax patients to cough while the wound was open, thinking they could cough out air. He thinks they can, but it will enter the next inspiration. Puncture may be indicated but the chest will fill soon.

The question of paracentesis is further discussed.

It is fifteen years since the appearance of his book. Either his opinions have changed somewhat or these lectures were poorly reported.

117. The Coin Sound. Pneumo-thorax. Nouveau signe physique pathognomonique de cette affection. *Gaz. des Hôp.*, Paris, Apr. 4, 1857.

After describing a case, a writer says a sign was present which was discovered and reported several years before by Professor Trousseau, but which to that time had not attracted attention. It consisted in putting the ear against the posterior chest wall of the patient while somebody percusses the anterior wall, either using a plessimeter and hammer, as

the professor was accustomed to do, or more simply using two pieces of money as plessimeter and hammer, or a coin and the finger. There is heard a metallic sound very sharp and resounding and often of such intensity that the ear is, as it were, wounded . . . It is a sound exactly like that made by striking a sharp blow on a brass vessel. This sound is pathognomonic of air in the pleural cavity. It cannot be mistaken, and occurs only in pneumothorax.

118. Clark. Pneumothorax. *New York Med. and Surg. Jour.*, Jan., 1859.

This was a case of pneumothorax developing early in tuberculosis.

119. Parks. Pneumothorax. *Boston Med. and Surg. Jour.*, Vol. LX, p. 501, July 21, 1859.

The case reported was that of a girl, 12 yrs. old, with tuberculous pneumothorax, in which the metallic tinkling was particularly loud, which Parks ascribed, since the perforation was not below the level of the fluid, to the explosion of mucus bubbles at the orifice. **Bigelow**, in the discussion, held that the perforation in question could have been submerged, and that in nine cases out of ten the tinkling is produced by the entrance of bubbles below the surface of the fluid.

120. Thornburn. Case of Idiopathic Pneumothorax, with Remarkable Absence of Bad Symptoms, and Recovery. *Brit. Med. Jour.*, 1860, I, p. 413.

Case 1.—A man, 22 years old, had for the past few days taken more exercise (rowing) than usual. He was awakened from sound sleep by a sharp pain in the right side and dyspnœa, lasting 2 or 3 hours. The next day he went to business but in the afternoon consulted the writer for pain in the chest and dyspnœa; pleurodynia was suspected.

On the third day the diagnosis of complete right-sided pneumothorax was made. The symptoms were very slight. There was no fluid exudate. In seven months he was well.

Thornburn considered there might have been a minute extravasation of blood on the surface of the lung, the result of rowing, and that this afterwards disintegrated. He knew of no previous case. He emphasized the latency of the trouble.

121. Biermer. Zur Heilungsgeschichte und Diagnose des Pneumothorax. *Würzberger medic. Zeitschr.*, Vol. I, p. 384, 1860.

It is now well recognized that pneumothorax per se is not very dangerous, and Wintrich et alt. have shown that air is not a severe inflammatory agent in the pleura. But if the condition be superimposed on a disease, the matter is not so simple, and hence the healing of a non-traumatic pneumothorax is exceedingly rare. Lebert (*Handbuch der prakt. Med.*, Tübingen, 1859, II Bd., p. 177), doubts the diagnosis in those cases reported as cured, but thinks that cases of pleural effusion were thus diag-

nosed. The writer saw one case which he now publishes at length. A boy, 19 yrs. old, at a dance, had a sensation as if his heart moved from its place. Other symptoms followed. The history and physical signs of this case of pneumothorax are minutely given. The patient recovered from the pneumothorax. Biermer then discusses the diagnosis of this case:

1. The sudden onset with the sensation of disturbance of the thoracic viscera, speak for pneumothorax rather than pleurisy.

2. The considerable displacement of the heart to the right was more than in pleurisy.

3. Movable dulness.

4. For the metallic phenomena pneumothorax, a cavity, or the larger bronchi are necessary, and here the latter two are not sufficient to explain this case, and pleural effusion does not produce signs of this degree.

He now reviews the literature of such phenomena in pleurisy and pneumonia. Williams first showed that in these two diseases amphoric phenomena do occur. In two cases of pleurisy he found over the exudate an amphoric percussion note and amphoric respiration, hence at first made the diagnosis of pneumothorax. He considered these sounds to be produced in the trachea and larger bronchi, and transmitted through the compressed lung.

Skoda did not recognize Williams' tracheal tone, and Wintrich defends the latter; Biermer agrees with Wintrich, but does not consider compression or hepatization of the lung necessary to produce the metallic tone, and cites two cases of Notta. He discusses the opinions of Behier, Beauvais, Landouzy, Friedreich, Hirsch, and Skoda (references are given). From the above reviews, he says, it is evident that for many the amphoric metallic phenomena have lost their value, but, Biermer says, unjustly, for their observations are not extensive enough. One must admit that in rare cases of pneumonia and pleurisy the Williams' tracheal tone (tympanitic or even with a metallic ring) is heard. It further cannot be doubted that a tubular respiratory murmur with an amphoric character does occur in some cases where there is neither a cavity nor pneumothorax. But the case is different as regards the metallic phenomena, for this is not claimed justly in cases of pneumonia or pleurisy by any of the above writers, and Skoda put the weight of his authority against it. These occur in pneumothorax cavities and bronchiectasis. The amphoric and metallic signs in pleurisy and pneumonia, he thinks, must be different. The amphoric breathing from the trachea, bronchi or throat is (1) clearest at the root and apex of the lung; (2) it is neither so loud nor exquisite as in pneumothorax or a cavity, and diminishes or disappears when the person breaths softly. The metallic tinkling is most valuable when not in the interscapular or infraclavicular region. Wintrich's change of pitch receives minute attention, but no very positive opinion is expressed.

The casuistry of pneumothorax is now discussed. The literature is well reviewed.

122. Noble. Some Particulars of Treatment in a Case of Pneumothorax. *Brit. Med. Jour.*, June, 1860.

This is a case of spontaneous pneumothorax. There was no suspicion of pulmonary disease before or four years after the attack. The patient took unusual exercise one day and the following morning while breakfasting the pneumothorax suddenly developed. Recovery in two months.

123. Biederhoff. Pneumothorax. Bonn, 1861.

Two cases from Naumann's clinic are reported.

The following thesis is of particular interest as it is the first critical review of the cases of a much debated form of pneumothorax.

124. Proust. Du pneumothorax essentiel ou pneumothorax sans perforation. Paris, 1862.

We will begin by giving his conclusions:

1. Idiopathic pneumothorax, or the secretion of air into the pleura, is contrary to the generally accepted facts of physiology.

2. No observation published until that time under that name appears to him to have been the object of an exact interpretation.

3. All such cases may be variously explained.

Some are pneumothorax in name only.

Some were cases of pneumothorax with or without the Skodaic note, with or without the amphoric resonance.

Some were cases of pneumonia at that stage when the percussion tone is sometimes tympanitic.

Some were symptomatic pneumothorax, the result of the rupture into the pleura of a tubercle or hydatid cyst.

Others seem to have been due to the rupture of a pulmonary lobule, resulting from an effort.

Such are the conclusions of a student the title page of whose thesis bears the names of Bouillaud, Rostan, Piorry and Trousseau as Professors of Medicine.

He considers that air long in contact with the pleura will inflame it, and sooner or later will cause a liquid exudate, except in some rare cases.

In his thesis he considers only Laennec's first two varieties:

1. Simple gaseous exhalation.

2. Gaseous exhalation complicating a fluid exudate but without putrefaction.

The possible sources of the air in such cases is the external air, the air in the lungs, the gases of the blood. He reviews the physiology of respiration and the physics of the chest, reviews the physiological arguments against essential pneumothorax, and next turns to the clinical evidence. In literature he finds twenty-five cases thus reported.

1. Some are pneumothorax in name only.

Cases I-II. Venot's cases.

2. Some were cases of abscess of the lung.

III. Ponteau (*Œuvres posthum.*, Vol. I, p. 341).

IV. Littré, *Mem. de l'acad. de Sc.*, 1713, p. 10.

V. Meckel's case.

3. Some are cases of pleurisy.

VI. Saussier's case (Legrous), in which at autopsy no air could be heard to escape from the chest but the space found could have been filled only by air. The lung was airless.

The Skodaic resonance deceived many. Roger found this present in forty-one of fifty-one cases of pleurisy.

VII. McDowel was thus deceived, who diagnosed it in a case without Hippocratic succussion, with no metallic tinkling, no amphoric respiration, which recovered.

4. Some were confused by the amphoric resonance.

Behier, 1854. Also Rilliet and Barthez, 1853; Landouzy, 1856; Beauvais, 1855.

These showed that amphoric respiration, which if considered alone would lead one to enough to diagnose air could exist in pleurisy. On this ground he condemns VIII, Saussier's case 10; IX, Maréchal.

5. Cases of pneumonia were mistaken.

Graves, Stokes, Hudson. Durrant's cases were condemned.

6. In other cases the perforation was overlooked.

Laennec, case XXXVII; Andral; Gintrac.

7. Others were cases of rupture of the pulmonary vesicles due to exertion; e. g., case of Ferrari, Combalusier, Ricker, Ranking.

There was doubtless great need of such a thesis as this to call attention to the danger of diagnosing pneumothorax on too small evidence, and we may be sure that the seven mistakes he mentions were made and are made, but one must admit that Proust was too critical, and has condemned cases which so far as the written evidence goes may well have been pneumothorax. It is very hard to deny that a person saw or heard that which he states he saw and heard.

124a. Rosenthal (quoted from Zuppinger). *Wien. med. Halle*, 1862.

A 16 yr. old boy swallowed a fragment of bone, which stuck in the œsophagus, and perforating into the pleura caused pneumothorax.

125. Pollock. Perforation of the Lung, Pneumothorax and Effusion into the Pleura from the Bursting of two small Tuberculous Cavities, in a Child three and a half years old. *Trans. of the Path. Soc. of Lond.*, Vol. XIV, p. 17, 1863.

The tuberculosis had been unsuspected (although there were slight symptoms). He died on the third day. Autopsy.

126. Leconte and Demarquay. The Gas in Hydropneumothorax. *Gaz. méd. de Paris*, Vol. 18, 3rd s., p. 114, 1863.

These writers have studied the gas in the tissues of animals and find it the same as in man.

In a case of emphysema there was found O, 6 per cent; CO_2, 5 per cent; N, 89 per cent.

The gas was analyzed which was obtained from a case of hydropneumothorax.

	1st portion.	2nd portion.
O	1.540	5.392
CO_2	10.820	8.823
N	87.640	85.785

Four days later:

	1st portion.	2nd portion.	3rd portion.	4th portion.
O	0.49	5.42	9.45	15.37
CO_2	11.76	9.36	7.96	1.53
N	88.35	85.22	82.59	83.10

Eight days later:

	Before removing 1 l. fluid.	After.
O	0.91	18.86
CO_2	10.55	1.88
N	88.54	79.26

These figures are interesting since they are claimed as the first analyses of the gas from hydropneumothorax of a living man (but see Davy), and since the gas differs from that in emphysema. The increase in O during aspiration is good diagnostic proof of the patency of the perforation.

Cl. Bernard found that of the gas in the arterial blood, $CO_2:O::9.12:100$; and of the gas in the venous blood, $CO_2:O::25:100$.

The authors found that of the gas in the emphysema, $CO_2:O::83.33:100$; and of the gas in the hydropneumothorax, $CO_2:O::1554:100$.

A comparison of these figures will show how great an alteration the gas in the pleura has experienced.

127. Corrigan. Pneumothorax. *Dub. Quart. Jour. of the Med. Sc.*, Vol. 40, 1865.

The case reported was one of tuberculous pneumothorax, with sudden onset, and extremely loud amphoric breathing. At autopsy air rushed out with such force as to blow out a candle. The fistulous opening admitted a small straw.

127a. Gérard. Causes, signes, et terminaisons du pneumothorax. Paris, 1865.

Causes.—A. Traumatic.

1. Fractured rib.

2. Contusion opening pleura: at once, or later following gangrene of chest wall.

3. Tear in pleura, violent compression. Gosselin said glottis must be

closed. It is only seen in young, and hæmatopneumothorax is always present.

4. Penetrating wounds.

5. Foreign bodies in the air passages. Leclère (*Thèse*, 1863. *Des perforations pulmonaires.*)

B. Nontraumatic.

1. Tuberculosis.

2. Pleurisy. Empyema expectorated through lung. Grisolle said this occurs in the encysted variety, and that air enters several days after the pus escapes.

3. Gangrene.

4. Emphysema.

5. Abscess of the lung. He cites Trousseau's case (*Clin. méd. de l'Hôtel Dieu*, t. I, p. 725). This form occurs especially after the pneumonia of aged or of children, and in children especially the pneumonia after measles. In this group there is no time for the formation of adhesions, and the only question is why it does not more often occur. There are no cases on record due to the common metastatic abscesses. He gives as references: Rilliet et Barthez, *Maladies des Enfants*, t. I, p. 427; Barrier, *Maladies de l'Enfance*, t. I, p. 363; Maréchal, *Jour. hebd.*, t. 11, 1829.

6. Pulmonary apoplexy is a very rare cause. Genest showed that infarct caused gangrene, and this the pneumothorax.

7. Hydatid cysts of the lungs. Cases of Mercier, Fouquier and Saussier.

8. Hydatids of the liver. Cases of Gros, Fouquier, Williams. See Lemaire (*Thèse*, 1837).

9. Cancer of the lung. Andral's case.

10. Abscess of the bronchial glands. Rilliet and Barthez are quoted. This is a rare form, since a thick fibrous coat forms around the glands.

11. Cancer of the stomach. Andral (*Clinique méd.*, t. II, p. 64).

12. Perforation of a bronchus. Andral (t. IV).

13. Rupture of the œsophagus.

C. Essential Pneumothorax.

Clinical Occurrence.

Sex.

Age. For this disease in children, see Bouchut (*Traité des mal. de l'Enfance*, p. 360).

Gas.

Symptoms.

Local. Pain at onset. The cause is not alone the pleurisy, but the tear of the lung.

Dyspnœa. This is absent in some cases.

Physical Signs.

Distension.—Lemaire (*Thèse*, 1837) showed the chest looked larger than it measured, hence inspection more valuable than mensuration.

The Fremitus is more often diminished than absent, except over the fluid.

The PERCUSSION TONE, Skoda and Hughes say, is dulled as tension increases.

In the above review of the subject only additional points are here given.

128. Weber. DE PNEUMOTHORACE. Berlin, 1865.

This contains the report of one case, otherwise nothing new.

129. Demarquay. ESSAY ON PNEUMATOLOGY. 1866.

PART 2, CHAPTER I. EMPHYSEMA IN GENERAL.—The force producing the emphysema emanates from the wounded organs or the organs accessory to it, e. g. in the case of a fractured rib wounding the lung the emphysema is produced by the functioning of the respiratory apparatus. At each inspiration the air enters the bronchi and finding an exit in the wounds of the lung passes into the cellular tissue of the thoracic wall. It escapes in greater amount if the patient makes any effort, closing the glottis, contracting the chest and pressing more air along the line of least resistance, i. e. the tear in the lung. The force producing the emphysema may be vis a tergo or aspiration.

CHAP. II. EMPHYSEMA IN PARTICULAR.—*From lesions of the larynx and trachea.*—Here the writer puts the cases of emphysema caused by the efforts of labor. There are but seven cases on record, all cases of primaparæ with difficult labor. The position of the tear has never been determined, since no case has come to autopsy, but since the emphysema appears on one side of the neck rather than bilaterally, the author thinks the tear is high.

From lesions of the bronchi and lungs.—a. *Following rupture of pulmonary cavities in phthisis.* Usually pneumothorax results, but if the ruptured part is adherent the perforation will involve the chest wall and emphysema result.

b. *Following the rupture of bronchi and pulmonary vesicles* due to paroxysms of coughing. In this convulsive movement we have first deep inspiration, then closure of the glottis and then violent expiration. As a rule the glottis opens, relieving the tension, but the rupture follows in some cases, of labor perhaps, after violent efforts, in the convulsions of hydrophobia, and from convulsive coughs. The visceral pleura always remains intact, the air accumulating under it forming bullæ. It then dissects its way along to the mediastinum. If the rupture in the lung is deep the air works its way along the bronchi and vessels, then up along the trachea to the neck. Or it may work its way along the œsophagus to the abdominal wall. From the neck it spreads over the chest and face, rarely further. It is a rare condition, most common in children about two and a half years old, more rare in adults. It is hard to explain this, since one would expect the tissue of a child to be more elastic. The author thinks the relative muscular weakness of adults saves their lungs.

c. *Following rupture of the lung without injury to the thoracic cage.*—A rare form, since the occurrence of several factors is necessary. Hewson's, Smith's and Gosselius' cases are given as illustrations. This occurs

in the young, since great flexibility of the ribs and cartilages is necessary. Violence must be applied to the chest while the person is making an effort immobilizing the chest, closing the glottis, the lungs being distended by considerable imprisoned air. The violence applied to the chest then (by a run-over accident, compression, etc.) will cause the enclosed air to rupture the lung. If the pleura ruptures, hæmopneumothorax results; if not, emphysema.

2. *Emphysema from lesion of the external respiratory apparatus.*

a. *Wound of the chest.*

(1) Simple wounds without lesion of the parietal pleura, the air being imprisoned by the respiratory movements.

(2) From penetrating wounds opening the pleura but not injuring the lung. This form of emphysema is excessively rare, if it ever occurs. As a result of such wounds the lung would be expected to collapse because of its elasticity, but it does not. The lung remains against the chest wall, and if pushed away will return at the next expiration, expelling the air which has already entered, unless the lung be pushed in considerably and enough air enters to cause pneumothorax. (The experiments of Dolbeau are cited as proof of this statement.) If the opening is large the air enters and leaves freely, but if it be oblique and narrow air is pumped into the subcutaneous tissue. As a matter of fact such a case of emphysema is rare, if it ever has occurred.

(3) Emphysema consequent to penetrating wounds of the chest with wound of the lungs.

Petit's theory is generally accepted. There is a certain amount of pneumothorax preceding the emphysema and the air in expiration must escape by the parietal wound causing emphysema if the skin wound be difficult to pass. Roux and Richet considered adhesions necessary, since otherwise the two wounds would not remain in contact. Yet the adhesions need not be present (see Littré's case). The percussion note may deceive in such cases and pneumothorax be suspected when absent. Emphysema of the chest means nearly always a wound of the lungs. The dyspnœa in such cases the writer considered due chiefly to the embarrassment of respiration caused by the effect of emphysema on these muscles, hence the much-recommended incision to relieve such cases he would reserve for extreme cases, and Hewson's opinion that the compression of the lungs by the air enclosed is the cause of the dyspnœa he says is based on a false theory. Such dyspnœa is also present when the collapse of the lung is prevented by adhesions.

Malgaigne, Sur le traitement des grands emphysèmes (*Bull. de Thérapeutique*, 1842), was the first to refute Hewson's view. Demarquay advises punctures and scarifications.

Emphysema following fractures of the ribs and sternum.—In the case of fractured ribs emphysema is a common complication. The first case was reported by Paré. The greatest emphysema arises when the rib fragment remains implanted in the lung, holding the lung against the chest wall and the parietal wound open. (See Méry's case, 1713.) If the lung is

free, with inspiration air is pumped into the chest, with expiration into the tissues. The lungs may collapse either partially or completely. Much emphysema is uncommon.

CHAP. III. PNEUMOTHORAX.—Nature of the gas in the pleural cavity.

There is no evidence of essential pneumothorax resulting from nervous disturbances or from gases of the blood.

Modifications which the gas undergoes. The absorption of O and increase in CO_2 renders the mixture more absorbable than is air. It is a sort of heterotropic respiration. (The findings of Demarquay and Leconte.)

The action of air on the pleura.—1. Chemical. Some think it has no effect, some think much. Demarquay takes an intermediate position. If the air is in contact with the pleura for a long time and is constantly renewed through an open fistula it may be supposed to cause a pleurisy more or less intense. If, however, its composition is modified and it is rapidly absorbed, the pleura is not affected. As proof of this he calls attention to the fact that in a case of pneumothorax supervening upon a case of tuberculosis there is an exacerbation of the thoracic symptoms. If the air is noxious to the pleura this is due to the oxygen contained, contrary to the opinion of Martin-Solon. If, however, H_2S or $(NH_4)_2S$ are present the gas is toxic, yet such cases are rare (Saussier found the gas fetid in 19 of 147 cases).

2. Mechanical. Slow asphyxia may result from the compression of the lung. The effect of the dislocation of other organs is discussed.

In conclusion he decides that the effect of the air is benign if in small amount; the more normal the pleura the better can it absorb the air.

The writer then tried animals. Air was injected into the chest of rabbits and pleurisy or even pneumonia resulted, but since the susceptibility of the animal may be at fault dogs were tried, air being injected every two days for weeks and no pleurisy was the result, hence he decides that air does no injury.

130. Roe. CASE OF LEFT PNEUMOTHORAX FROM AN OPENING IN THE RIGHT LUNG. *Med. Times and Gazette*, Apr. 7, 1866.

A man, 32 yrs. of age, tuberculous, was suddenly seized with pain and dyspnœa. Left-sided pneumothorax was found. At autopsy the current of air which escaped from the wound blew out a match. The pneumothorax was on the left side, but the perforation was on the right, that lung being very adherent. (The opening between the right lung and left pleural cavity was not demonstrated, since the lungs were first removed before inflation was tried.)

131. Vogel. RASCHE GENESUNG VON EINEM PNEUMOTHORAX. *Deuts. Arch. f. klin. Med.*, Vol. II, p. 244, Sept. 7, 1866.

The case was that of a woman, apparently healthy, 29 yrs. old, whose right-sided pneumothorax developed "like a flash of lightning from a clear sky" and who was well in about four weeks. There was no pleural effusion.

Biermer's claim that the pitch of the metallic tinkling depends on the position of the patient was not confirmed in this case.

132. Russell. Case of Pneumothorax occurring at the very Commencement of Tubercular Deposit of the Lung. *Brit. Med. Jour.*, Mar. 24, 1866.

The patient was a man 33 yrs. old. He had had slight pulmonary symptoms for about five months. He could not state the time of onset of the right-sided pneumothorax. He died in from 4 to 5 weeks, and the autopsy confirmed the diagnosis. The tuberculosis was slight.

The early appearance of this complication and its latency are the points to which attention is called.

133. Skoda. Pneumothorax (A Clinical Lecture). *Wien. medizin. Presse*, 1866, VII, pp. 1202, 1232, 1256.

Hasty examination discovered nothing new in this lecture. It seemed much the same that this author has elsewhere written.

134. Abeille. (Extensive Pneumothorax of the Right Side. Recovery.) *Gaz. méd. de Paris*, 1867.

The case is reported of a man 29 yrs. old, in excellent health, who was awakened one morning by the sensation of cold air penetrating the right side of the chest, followed by violent pain at the inferior angle of the right scapula. There is history of a strain the evening before. He recovered without fever or fluid. Metallic tinkling was present with each inspiration, although fluid was absent. It is considered as a case due to emphysema.

135. Curling. Case of Traumatic Pneumothorax. Paracentesis. Recovery. *Med. Times and Gazette*, Oct. 26, 1867.

A boy 13 yrs. old was run over by a cab, causing left-sided pneumothorax. No fractured rib was discovered.

135a. Guttmann. A Contribution to the Physiology and Pathology of Respiration. *Virchow Arch.*, Vol. 39, 1867.

The writer first reviews the observations made of respiration after the vagi have been cut (quoting Wundt). Cutting the vagi results in slower deeper breaths, yet (excepting in the case of birds) the amount of air respired per 1 min. (Athmungsgrösse) is nearly unchanged (Rosenthal). But the Athmungsgrösse later becomes less, due to changes in the lung diminishing the respiratory surface, and exhaustion of the respiratory muscles. But at first the Athmungsgrösse may be even twice the normal.

He describes his experiments, using Hutchinson's spirometer, confirming the above statements, and then asks, "if the vagus physiologically has no control over the Athmungsgrösse . . . does it exert an influence under pathological conditions, i. e. in dyspnœa," but his experiments on this point failed.

The next point considered is the effect on the Athmungsgrösse of diminishing the respiratory surface, by the injection of fluid into the pleura. In pleurisy during the exudation dyspnœa is seen, but when the febrile period ceases, with the chest full of fluid, the respiration is often quiet.

1. Experiments with the injection of fluids into the pleural cavity. Rabbits were used. Dyspnœa occurred only after considerable had been introduced.

2. Also in pneumothorax the increased exertion of respiration had as little influence on Athmungsgrösse as in hydrothorax.

136. Powell. Lung showing Perforation, from a Case of Pneumothorax. *Trans. of the Path. Soc. of London*, 1868.

At autopsy, the pressure in the chest was 4 in. H_2O. The opening in the lung (tuberculous) had a most perfect valve of the pleura. The interest in the case consisted "in its illustrating the relation between a very perfect valvular opening found after death and the absence of amphoric or of any respiration, and the steadily increasing dyspnœa which indicated it during life." These two symptoms indicate a valvular opening and show necessity of paracentesis. The determination of air pressure during life is advised, both in pneumothorax and fluid effusions, "the pressure in both of which have as yet been ascertained in a very few cases."

("Powell," said Bard, "was the first to demonstrate the positive pressure in the chest in valvular pneumothorax.")

137. Fairfax. A Case of Pneumothorax with Metallic Tinkling. *Richmond Med. Jour.*, Feb., 1868.

In this case the "metallic tinkling was so loud as not only to be perceived and very accurately described by the patient, but to produce extreme alarm and distress in her mind." The case was one of tuberculous pneumothorax. At the first visit the patient said: "'Oh! doctor, what is this dropping inside my chest? It will distract me." She said it was "exactly like water dropping, dropping, on a sheet of tin." The well-marked metallic tinkling was synchronous with respiration, and would continue for perhaps a dozen drops and then cease, to be renewed upon the patient's coughing or speaking. "The right back was tympanitic on percussion down to the false ribs. I found more respiratory sound in this part than I suspected, but it had lost its vesicular character and had become decidedly amphoric. . . . She died that night. No autopsy could be procured."

This is one of the rare cases in which metallic tinkling is reported as audible to the patient.

138. Powell. Notes on the Pneumothorax occurring in Phthisis. *Med. Times and Gazette*, Jan. 30, 1869, and Feb. 13, 1869.

Pneumothorax may arise in phthisis from the softening of a tubercle before adhesions have protected the pleura, or by sinus formation from an old cavity to the pleura.

After discussing symptoms and physical signs Powell considers the question concerning the displacement of the heart, which with hyperresonance and the auscultatory features of respiration constitute the three most constant symptoms of this disease. The cause of the displacement, he was the first to prove, is not positive pressure, but a diminution of the negative tension on one side, hence the heart, balanced between two negative tensions, will be "drawn" to the normal side. This was shown on the cadaver and on a dog, a needle being inserted through the chest wall into the heart; if one side of the chest was then opened the heart moved at once to the other side.

The intensity of the symptoms in pneumothorax is influenced by:

A. The condition of the lung previous to its rupture, the symptoms being more marked if the lung were normal. To be considered are:

1. The amount of respiratory area cut off.

2. The increased resistance to the circulation in that lung.

Powell now discusses the effect of positive pressure in one pleural cavity on the circulation of the collapsed lung. Goodwyn, 1788 and Erichsen, 1845, had shown that mere collapse of the lung did not materially affect the circulation through it.

The actual pressures were determined in eight cases at autopsy and they varied from $1\frac{1}{2}$-$5\frac{3}{10}$ inches of water. An artificial thorax was arranged, dog's lungs being put in an air-tight vessel and the effect of various pressures tried on its artificial circulation. His conclusions are "That there is no appreciable difference in resistance to the circulation through a moderately expanded lung, and one collapsed by its own elasticity; that when the lung is more completely collapsed there is a decided increase in the resistance through it; that the complete collapse of the lung has a very marked effect in retarding the circulation through it, independently of all external pressure."

B. The state of the other lung, which will have less elastic tension, hence will be hampered by the displaced heart and, if present, by direct pressure.

The effect of positive pressure on the circulation to the heart and other points are considered.

Ibid., Feb. 20, 1869: A brief history of each of the eight cases of pneumothorax whose intrapleural pressure was measured is given.

139. Cotton and Alison. Cases Illustrating the Manner in which the Heart is Displaced in Pneumothorax. *The Med. Times and Gazette*, Aug. 21, 1869.

These two cases were studied with reference to the point Powell had made, that the displacement of the heart is not the result of a positive pressure. At autopsy the intrapleural pressure of the pneumothorax side (the right) was tested and found to be 0. The position of the heart was then determined and in each case the apex was found behind the sternum. Hence extreme displacement of the heart is no sign of intrapleural pressure.

It will be noted that in these cases the pressure was determined only after death.

140. Anstie. Pneumothorax. *Reynolds' System of Medicine*, 1871.

This brief review of the subject is of interest only in that he classifies the subject into two varieties:

1. Non-perforative, due to—

a. Decomposition in gangrene of the pleura.

b. Decomposition of an ordinary pleuritic fluid.

c. The formation of a gaseous exudate, replacing a sero-purulent fluid suddenly absorbed.

d. Secretion of air by the pleura.

2. Perforative, with many subdivisions.

(It is interesting that he accepts these non-perforative forms.)

He states that the percussion note becomes more muffled as the tension of the gas increases, "a well-known phenomenon of extreme air tension."

141. Ramskill (or) Mackenzie. Two Cases of Pneumothorax treated by Aspiration. *Lancet*, Aug. 19, 1871.

(These cases were reported by Mackenzie, but were under the care of Dr. Ramskill. Both names have been used in referring to them.)

Case 1.—A man of 50, whose pneumothorax was probably due to the rupture of an emphysematous air vesicle, was greatly relieved by aspiration. He recovered.

Case 2 was a woman 28 yrs. old, a tuberculous case of pneumothorax, who experienced great relief from aspiration.

The writer recommends aspiration as a trivial, painless operation, with no drawbacks, and which gives great relief.

142. Thompson. Case of Pneumothorax. *Lancet*, Sept. 16, 1871.

The patient was a man 52 yrs. old, who had for six months had a persistent cough, and who was admitted evidently for consumption. At autopsy pneumothorax was discovered.

In discussing pneumothorax in general Thompson uses the term *centripetal*, as in rupture of an empyema into the lung causing pneumothorax, and *centrifugal*, as occurs in tuberculosis. He considers his a case of the latter, although he thinks the vomicæ, one of which ruptured causing the pneumothorax, of pneumonic, rather than of pyemic or tuberculous origin.

He discusses the diagnosis in such cases.

143. Thompson. Case of Hydropneumothorax. *Brit. Med. Jour.*, Oct. 5, 1872.

The final report of a case, J. B., aged 22 yrs., of typical "hydro" pneumothorax of tuberculous origin, and death due to "drowning in pus." Autopsy.

144. Noble. Some Particulars of Treatment in a Case of Pneumothorax. *Brit. Med. Jour.*, Oct. 11, 1873.

A man, 40 yrs. old, previously in good health, awoke one night with acute violent pain in the right side, which lasted several hours and was fol-

lowed by some difficulty in breathing. Seven days later the diagnosis of pneumothorax was made. Paracentesis was twice performed with good results. In this case a rubber tube was attached to the canula and its end put under water, that the bubbling might demonstrate the presence of air. "Air and air only" escaped. The patient soon recovered.

145. Sturges. Air Entering the Pleura, not from the Lung; Hydropneumothorax; Partial Recovery. *Lancet*, Feb. 7, 1874.

A soldier, 28 yrs. old, who had suffered from daily vomiting ("chronic dyspepsia") for the past six months, and for the past seven months cough, slight expectoration, and who on one occasion had spat up blood, had when walking a sudden attack of chill, catching pain in his left side, and considerable dyspnœa. The symptoms of pneumothorax on the left side, including, later, succussion, were definite. The man was recovering at the date of report.

(The author is inclined to consider the cause of the pneumothorax a gastric ulcer perforating through the diaphragm, but the symptoms of ulcer are to our mind not so marked as those of tuberculosis of the lung which Sturges thinks he can exclude. These were chronic cough with expectoration, on one occasion slight hæmoptysis, persistent vomiting, an old pleurisy and physical disability from these sufficient to keep him a hospital patient.)

146. Da Costa. On the Terminations of Pneumothorax; Especially on its Termination in Recovery. *Phila. Med. Times*, June 20, 1874.

The lecturer presented one and described other cases, illustrating these three modes of termination:

1. Perfect recovery, not a rare mode.
2. Cure of the pneumothorax, but the chronic pleurisy remaining.
3. Persistence of the pneumothorax, "chronic pneumothorax," but the patient remaining in fair health for some time.

147. Cole. A Case of Fistulous Pneumothorax. *Lancet*, Sept., 1874, II, p. 340.

This was a case of tuberculous pneumothorax, in which in the lower posterior axilla was heard a remarkable amphoric note. At autopsy was found at this point the wide-open fistula into the bronchus.

148. Wilks. Simple Pneumothorax, with Complete Recovery. *Brit. Med. Jour.*, Dec. 19, 1874.

Case 1 was of a bill sticker, who while at work fell to the ground because of left-sided pneumothorax. He was brought to the hospital in an apparently dying state, but speedily recovered, and no pleural or pulmonary disease was found.

Case 2 was an elderly man, in which probably an emphysematous bleb ruptured. Recovery.

Case 3.—Woman, 30 yrs. old, who caught cold and ten days later had left-sided pneumothorax, which suddenly developed. Recovery.

149. Gairdner. Pneumothorax. *Lancet*, 1875, I, 605.

The case of a boy, 13 yrs. old, who, following a pleurisy showed the symptoms of "a cavity partially air-filled in the right pleura corresponding with the position of the middle lobe and circumscribed more or less by adhesions over the upper and back part of the lung" is reported at length. The boy recovered. Gairdner considers the case must have been tuberculous. In the discussion following the writer considers the conservative power of the adhesions, agreeing with Alison (*The Physical Exam. of the Chest in Pulm. Consumption*, London, 1861, p. 270) that "it is a strictly correct statement to say that pneumothorax tends to occur in every case of tuberculous disease, and that in almost all cases which reach the stage of excavation it would inevitably occur were it not for the presence of adhesions."

149a. Müller. Pneumothorax from Gastric Perforation. Round Worms found in the Pleura. (Quoted from Zuppinger.) *Schmidt's Jahrbuch*, 1875.

A 16-yr. old girl developed pneumothorax on the left side and died in six days. In the pleural cavity were found three round worms which had entered through a perforation from the stomach into the pleura.

150. Butlin. Notes on Three Cases of Air in the Pleura as a Result of Injury. *St. Barth. Hosp. Rep.*, Vol. XI, p. 255, 1875.

Case 1.—Left-sided pneumothorax in a man 22 yrs. old, due to a severe fall; later hydropneumothorax; no fracture of ribs or sternum detected. Recovery.

Case 2.—Left-sided pneumothorax in a child 6 yrs. old, run over by a heavy vehicle. No fractured bones detected. Well in 6 weeks.

Case 3.—A man caught between the bumpers of cars was severely injured, and gave the signs of left-sided pneumothorax. At autopsy the diaphragm was found ruptured and the pleural cavity contained the stomach, spleen and transverse colon.

151. Church. Notes on an Interesting Case of Pneumothorax. *Edinburgh Med. Jour.*, June, 1876.

This was the case of a "hammer-man," 37 yrs. old, who was taken suddenly ill, "brought home in a dying state," and found in a state of collapse, able to breathe neither in nor out. The right side was much distended and its note highly tympanitic, except posteriorly, where there was slight dulness. A trochar was inserted, air hissed out; the dislocation of the heart diminished, and relief followed almost immediately. After the air had escaped the percussion note was tympanitic, but not of so high a pitch. No pleurisy developed, no metallic tinkling was heard. Recovery in about three weeks.

Church thinks this a case due to the rupture of an emphysematous bleb.

152. Ewald. Untersuchungen zur Gasometrie der Transudate des Menschen. *Arch. f. Anat. und Phys.*, 1876, p. 422.

A lengthy review of the subject precedes. His technic is simple (see 155). If the CO_2 is + 10 per cent he is confident of a closed fistula.

The analyses of fifteen pneumothorax cases are given in per cent of CO_2, O and N (the O was absorbed by pyrogallic acid).

As a result of the study of the cases with purulent exudate he decides that the CO_2 tension of inflamed tissue producing a purulent exudate is between 15 and 20 volumes per cent, and the height depends on the severity of the inflammation. The oxygen tension of such tissue is about 2.9 per cent. The average N per cent was 79.81, hence he considers this gas indifferent.

In the case of serous exudate the CO_2 per cent is between 7.5 and 11.5.

Hence the CO_2 tension of the cells of inflamed tissue is in every case above that of normal blood, and may vary from about normal to even two or three times normal, depending on the duration and especially the intensity of the inflammation, and is highest in pus formation.

In this work the gases of the exudate were also determined (but these fluids were not removed always with the gas of the pleura, even several days before the gas, and in one case ten hours after death).

The whole subject is very well discussed.

153. Bernheim. Clinical and Therapeutic Considerations of Pneumothorax. *Lectures on Clinical Medicine*, p. 126, 1877.

After reporting a case of tuberculous pneumothorax the writer discusses the duration of the disease, the process of closure of the fistula, the time necessary for its closure (estimated by the duration of metallic tinkling), remarks on tapping, etc. Some unusual references are given.

154. West, S. Two Cases of Pneumothorax. *St. Barth. Hosp. Rep.*, Vol. XIII, p. 215, 1877.

Case 1.—Access marked by severe attack of pain. The patient was a girl 15 yrs. old, tuberculous, who was treated for incontrollable vomiting. Early one morning she awoke shrieking with pain in the left side, and pneumothorax. A cerebral tumor found at autopsy cleared up the reason for the vomiting.

Case 2.—A man 41 yrs. old, tuberculous, had pneumothorax on the left side, which was unsuspected before examination, as he had had no sudden onset, the only symptom being slowly developing dyspnœa.

155. Ewald. Ueber ein leichtes verfahren den Gasgehalt der Luft eines Pneumothorax und damit das verhalten der Perforationöffnung zu bestimmen. *Charité Annalen*, 1877 (1875), Berlin, Vol. II.

Ewald briefly reviews all former work on this subject and considers the results not comparable since various methods were used and the condition of the perforation not determined.

He considers this analysis the only certain means of diagnosing the condition of the fistula and has (152) tested its accuracy in 17 cases of pneumothorax, the result of the analysis being compared with the anatomi-

cal findings. He found he could separate two groups of cases: those with open fistula in which the O was 12-18 per cent, and the CO_2 5 per cent as a maximum, and those with a closed fistula with the O = 10 per cent as a maximum and the CO_2 15-20 per cent+. In addition were some intermediate cases. Hence if the CO_2 is under 5 per cent an open fistula is to be assumed; if from 5-10 per cent, a partial closure; and if + 10 per cent an encapsulated exudate. From 24 to 26 hrs. is long enough for the gas in the pleura to come to an equilibrium.

The apparatus he suggests is an inverted burette as an improvised eudiometer and a large syringe by which the gas is aspirated. The burette filled is inverted in a basin of salt solution. The gas is forced by the syringe into the burette and the amount read. Then a stick of KOH, pea-size, is introduced, the burette shaken, and the reading of the remaining gas taken. The difference is the volume of CO_2. By control tests he considers this method to be within from 0.5 to 1 per cent of accuracy.

He then reports a case in which all the clinical symptoms indicated closed pneumothorax, especially the marked dislocation of the organs, but the gas analysis (CO_2 = 3.3 per cent) indicated the open form, which was confirmed at autopsy. The writer discusses further the clinical discrepancies between pressure symptoms and the anatomical findings concerning the patency of the fistula.

156. Ruge. Pneumothorax bei einem Neugebornen. *Zeitsch. f. Geburtsh. u. Gyn.*, Vol. II, p. 31, 1877.

The child, in breech presentation, was easily extracted by the foot. It was asphyxiated at first but cried lustily that night. The next morning it suddenly became cyanotic and died. At autopsy, on opening the left chest, air hissed out. In that pleural cavity was a little blood. The lung was compressed, almost airless. The sequence seems to have been: violent respiration, causing alveolar then interstitial emphysema, then rupture of the pleura.

156a. Fräntzel. Ein Fall von doppelseitigem Pneumothorax. *Charité Ann.*, Oct. 11, 1877.

At autopsy double-sided pneumothorax was found. The left cavity contained two liters of fluid, the lung was fist-size and airless. The mediastinal tissue was very caseous and the perforation had occurred here.

Double pneumothorax is one of the rarest events of pathology. Puchelt (*Heidelberger med. Annal.*, Bd. VII) described one.

In this case the sequence was serous pleurisy, becoming purulent, and breaking through the left lung. Then tuberculosis of the mediastinum, which perforated into the right side. The patient survived the double pneumothorax for one and three-quarter hours, breathing with a zone of lung protected from collapse by the tuberculous thickening.

157. Garland. Pneumono-dynamics. New York. 1878.

Chapter XIV. Pneumothorax.—The principles involved in this condition are the same as in pleurisy with effusion. If air be admitted to the

pleural cavity the lung collapses by virtue of its elasticity, and will not be compressed except subsequent to complete collapse. The costal pleura of a dead dog was exposed over a wide area: a small hole was then made in it. The lung began at once to contract, the retraction being first manifested by a symmetrical elevation of the lower part of the lung. "The air collected in one body between the lung and diaphragm but no air, so far as I could see, penetrated between the lung and the lateral chest wall until the lower border of the lung had retracted upwards to the distance of several ribs. Finally, as the lower part of the lung continued its ascent, its lateral surface began to recede from the chest wall until a condition of complete collapse was established." The posterior part of the lung contracted with greater rapidity than the anterior part. The displacement of the neighboring organs is due to the absence of the negative tension on the affected side. This was also found to be the opinion of Skoda, and Powell, whose writings are quoted.

He thinks the heart is more displaced in pneumothorax than in pleurisy, because the air having practically no weight cannot exert upon the heart that negative pressure which an effusion evidently would.

This is from but one chapter of a book filled with new and suggestive ideas on the physics of the chest.

158. Desplats. HYDRO-PNEUMOTHORAX, THORACENTESIS TWICE PERFORMED. SEROUS EXUDATE ALSO AT AUTOPSY. *Jour. des. Sc. méd. de Lille*, Vol. I, p. 117, 1878-9.

A tuberculous patient, male, 31 yrs. old, with a cavity at the right apex, developed right-sided pneumothorax. To relieve his distress the chest was twice tapped and much gas and a transparent serous liquid escaped. The patient died and at autopsy similar fluid was found in the chest. "This interesting and rare case proves that the penetration of air into the pleura does not induce purulence so certainly as writers affirm. It shows also the harmlessness of aspiration puncture."

The fluid was analyzed. Precipitation with alcohol showed a soluble and an insoluble albumin, the rotation powers proved the same present before and after death (soluble albumin — 59.1, unsoluble — 68.6).

The author could find recorded but few similar cases. Saussier reported one case with serofibrinous exudate, Marais one, and Vieuille three. Peyrot (*sur la pleurotomie*) said he had seen one case of tuberculous pneumothorax, the fluid in which remained serous for several months despite repeated tappings. Landouzy had seen a similar case.

The writer concludes such cases are rare and none presented so markedly as his case those features supposed to favor the purulent transformation of the fluid.

Sudden Death caused by the Perforation of the Lung and the Sudden Production of Pneumothorax.—A tuberculous man, 20 yrs. old, developed suddenly left-sided pneumothorax, and died in a few hours. The lung was found entirely collapsed, about the size of the fist. There was no

trace of pleural adhesions, which the writer considers rare. A subpleural tubercle had ruptured.

Case of Pyopneumothorax limited to the anterior part of the chest and pushing the heart to the right.—A tuberculous boy, 19 years old, developed partial pyopneumothorax on the left side, anteriorly and inferior. Death. Autopsy.

159. Powell (R. Douglas). Clinical Lectures on Excavation of the Lung in Phthisis. *Lancet*, Feb. 2, 1878.

In this lecture Powell deals with pneumothorax as an accident that may happen to a tuberculous patient. He reports that he found five cases in more than one hundred autopsies on phthisical patients. Clinically, he reports for seventeen cases. He speaks of the early phthisis with subpleural tubercles, in which it is most likely to occur, of the symptoms and of the physical signs, and of these especially, the displacement of the heart which he considers a sensitive index of the presence and degree of pleural effusions. He illustrates this by an artificial thorax with bladders as lungs, showing the effect of the elasticity of the lungs.

"So in the living subject at the moment of the occurrence of pneumothorax and before any pressure recognizable by a manometer occurs a notable and instantaneous displacement of the heart takes place." "The clinical fact I want now to bring home to you from this demonstration is that displacement of the heart . . . is not necessarily a sign of intrathoracic pressure, and therefore, even when considerable, is not alone sufficient to call for relief by paracentesis."

160. Leichtenstern. Pneumothorax. *Gerhardt's Handbuch der Kinderkrankheiten*, Vol. 3, Part 2, page 962, 1878.

Etiology.—1. Inflammatory processes of the lung leading to ulceration of the pleura. This includes the most various chronic pulmonary diseases leading to cavity formation, etc. . . . the purulent or caseous lobular pneumonia in children, the result of the catarrhal pneumonia which so often follows measles; purulent or gangrenous pulmonary emboli; metastatic or other forms of lung abscess, lung gangrene.

2. Empyema rupturing through the lungs. (Page 896. In very many of these cases during the whole process of healing it does not result in demonstrable collection of gas in the pleural cavity, never in pyopneumothorax.)

3. Trauma, including those very rare cases occurring in whooping-cough. Penetrating wounds, etc.

Anatomical Findings.—He believes the pleura is filled in valvular cases by inspiration.

Symptoms.—Two cases are reported, one of sudden death at onset, one of latent pneumothorax. He emphasizes again the value of his "Plessimeter Stäbchen percussion" (the auscultation of the percussion on the plessimeter).

Under *Therapy* he advises the puncture of the thorax with a capillary trochar repeated as often as necessary.

For the most part this chapter would apply to pneumothorax of adults as well as of children. The latter phase of the question received slight attention.

160a. Traube. Zur Lehre vom Pneumothorax. *Gesammelte Beiträge,* 1878.

Case I was of tuberculous pneumothorax. The percussion note found on the autopsy table, over the much distended left side was very loud and abnormally deep, and without metallic resonance. This note, after opening the chest, had a slight metallic resonance. As a result of further observations Traube decided:

1. That a high grade of tension of the chest wall is unfavorable to the production of the metallic resonance.

2. That the tension of the gas in the pleura can be above atmospheric when but very little fluid is present.

3. The metallic resonance is louder when the compressed lung is under the level of the fluid than when wholly free.

4. The abnormal tension of the intercostal spaces can depend on the resistance which the abdominal organs offer to the expansion of the diaphragm.

Case II was an autopsy on a case of acute pneumothorax (the patient died at the onset). The right side was distended; the percussion note was louder and deeper without metallic resonance.

Case III is interesting since all signs of extreme distension were present, yet considerable pressure must be exerted on the chest to expel the air, showing its tension to be only slightly above atmospheric.

Page 349. In another case during life the most extreme distension was noted. The apex beat was three inches outside the left mammary line, the liver dulness began ¾ inch below the costal margin, yet when the trochar was inserted scarcely any air escaped. Traube explains this as due to a loss of elasticity of the distended thoracic walls. He made a similar observation in a case of pleural effusion. This patient was uncomfortable after a little air had escaped, and Traube decided in the future to compress the chest with a binder while allowing the air to escape.

161. Lyon. Pneumothorax with Embolism of the Pulmonary Artery. *Boston Med. and Surg. Jour.*, June 27, 1878.

The history of the case was presented before the Hartford County Medical Society. The clot found came probably from the left femoral and popliteal veins. "Air escaped on opening the chest." The lungs were tuberculous.

162. Cossy. Sur le pneumothorax engendré par des gaz provenant du tube digestif. *Arch. gén. de méd.*, 1879, II, page 526.

These cases may be divided into (1) those following perforation of the œsophagus; (2) of the stomach; (3) of the intestine. If the diaphragm does not perforate we have false pneumothorax.

Cossy reports two very rare cases of pneumothorax following perforation of the cœcum.

Case 1.—Man, 32 yrs. old, following perforation of the cœcum by a fisn bone, had peritonitis, perforation of the diaphragm and dissection of the parietal pleura from the chest wall.

Case 2.—A young man, 20 yrs. old, with "perityphlitis" followed by right-sided pleurisy. At autopsy the appendix was normal. A fistula running in the sheath of the rectus communicated with the cæcum and perforated the diaphragm. The cause of the fistula he does not know. The gas, however, was not in the pleura itself but in a cavity formed by the dissecting off of the parietal pleura.

Cossy, therefore, makes three groups of pneumothorax of intestinal origin:

1. That in which the gas is contained in the pleura itself.
2. That in which the gas occupies a space between the thoracic wall and the parietal pleura. Both of his cases were this type.
3. False pneumothorax (subphrenic).

163. Unverricht. Pneumothorax. *Zeitsch. f. klin. Med.*, Bd. 1, 1879,

This writer divided the cases sharply into:

"1. Cases with perfectly free communication between the lung and pleura. The lungs are in elastic equilibrium since the pressure in the pleural cavity is that of the atmosphere. All pressure symptoms fail. The slight dislocations of neighboring organs . . . are not due to pressure but to the elastic tension of the other lung and the diaphragm, hence these are better named 'retraction symptoms.'" Here operation is almost never indicated.

"2. Cases with closed fistula, which present more or less marked pressure symptoms, according as the exudate is considerable or not." If an operation is necessary simple aspiration is sufficient.

3. Cases of valvular fistula which may show such marked pressure symptoms that operation is imperative. These are the cases in which the need of accurate information concerning the fistula is imperative, since that will determine the operative procedure. If aspiration be done, the air will soon re-collect, but this time may be delayed if the patient avoid forced respirations, coughing, etc., by the use of morphin and other drugs.

164. West. Two Cases of Pneumothorax of the Right Side. *Trans. of the Path. Soc. of London*, 1879.

Case 1.—Tuberculous, male, 22 yrs. old; sudden onset relieved by tapping twice; death in forty-eight hours. On autopsy the liver was found remarkably twisted, its notch under the tip of the ninth left rib.

Case 2 was a man 40 yrs. old, with phthisis, with about the same history and anatomical findings as above.

"In the first case there was but litle caseous degeneraion, the changes in the lungs being almost exclusively those due to chronic bronchitis and

bronchiectasis, and that pneumothorax should develop under these conditions is, I believe, an event of rare occurrence."

165. Weil. Zum Lehre vom Pneumothorax. *Arch. f. klin. Med.*, Vol. XXV, p. 1, Dec. 10, 1879.

To Laennec and Wintrich he gives the credit of laying the foundations of our knowledge of the clinical side of the question. Biermer and Gerhardt have especially contributed to the symptomatology.

The clinical symptoms of a fresh pneumothorax are the results of the mechanical effect of the quantity and tension of the air in the pleura on circulation and respiration. These two factors (quantity and tension) control the symptoms, but to understand them the various kinds of pneumothorax must be distinguished.

A. Experimental Part.—Weil does not consider any cases in which the gas arose in the pleural cavity. He separates:

1. Closed pneumothorax, in which case the fistula allows no air to enter during either inspiration or expiration.

2. Open pneumothorax, in which cases the air has free entrance and exit during both.

3. Valvular pneumothorax, which is open on inspiration and closed during expiration. Soon the chest will be so filled that no more air can enter, and to such a case, although it is really a variety of the closed form, he gives the name valvular.

4. All transitions occur; for instance the smaller the fistula the nearer that open form approaches the closed.

Rabbit Experiments.—Water manometers, a Voit mercury valve, and a graduated cylinder filled with water which was allowed to flow into a Wolff flask connected with the pleural canula (which forced in and measured the air introduced), were used in producing the pneumothorax and studying the effects (a good picture of the apparatus is given). Nine rabbits were used.

The average intrapleural tension found was — 6.7 cm. H_2O, the average respiratory oscillation was 4.9 cm. H_2O. Air was now allowed to enter till the pressure at the end of expiration was 0. (This amount = "pleural volume.") This required on an average 17.5 cm. of air per 1 k. of body weight, and in the chest was a *closed pneumothorax.*

Average pressure on inspiration.....	— 7 cm.
Average pressure on expiration.....	+ 3 cm.
Mean pressure	— 2

In *valvular pneumothorax* the amount of air entering chest was 34.3 cc. per 1 k. The Voit valve used.

Average insp. pressure	— 1.1 cm.
Average exp. pressure	+ 5
Mean pressure	+ 1.9

In *open pneumothorax.* The chest opened.

Mean pressure	= 0
Average insp. pressure	— 0.4
Average exp. pressure	+ 0.4

That is, the oscillations were very slight.

Hence the mean pressure in closed pneumothorax is negative, in valvular is positive, in open is the atmospheric. The pressure oscillations are greatest in the closed and least in the open.

Changes in frequency of respiration and in the amount of excursion of the chest wall (using a Burdon-Sanderson Cardiograph) are now studied.

In closed pneumothorax the frequency increases on an average 35.5 per cent, and the excursion 25 per cent.

In valvular, frequency increases 17.8 per cent; excursion increases 54.4 per cent.

In open, frequency decreases 15.4 per cent; excursion increases with a wide fistula even five times, with a narrow fistula less.

(Very good tracings are given.) He could determine no difference in the excursions of the two sides.

He next asks the method by which in rabbits the different forms of pneumothorax affect the respiratory mechanism.

The space occupied by the air is made by the collapse of the lung, the enlargement of the chest, and the dislocation of the heart and diaphragm. In open pneumothorax the increased respiratory efforts can affect the collapsed lung very little since the intrapleural respiratory oscillation is lost, and does not aid the sound side much since the mediastinum is pulled still more to that side. As a result but about one-half the normal amount of air passes through the trachea.

In closed pneumothorax, at the end of expiration, we have almost the same conditions as in the open, but the pressure symptoms are more pronounced since in expiration the pressure is often positive. But the dyspnœa is very different, since frequency is increased and depth changed but slightly. But the intrapleural oscillation on the affected side is even 10 cm. H_2O, hence that lung can partake in respiration, and the amount of air respired be even greater than normal (excursion being greater and rate faster). To explain the slow, deep respiration of the open form he is not able.

In valvular pneumothorax similar conditions obtain to those in the closed form, but a larger amount of air is in the pleura, hence the pressure is positive and the lung compressed; that is, its "mean volume" is smaller than in the closed, and this in turn is larger than in the open, in which case it is equal to that of the retracted lung.

Dog Experiments.—The same experiments were performed on dogs narcotized with morphin.

He found that the conditions in open pneumothorax were similar, that the mean pressure in the closed form is more markedly negative, and in the valvular more positive. The pleural cavity of a dog is relatively twice

the size of a rabbit's, and in valvular pneumothorax the amount of air introduced relatively almost three times as great.

Normal.

Average insp. pressure	— 10.2 cm. H_2O
Average exp. pressure..... ..	— 4.6
Average mean pressure	— 8.5

Closed pneumothorax. Amount of air entering chest per 1 k. = 31.3 cm.

Average insp. pressure	— 7.3 cm.
Average exp. pressure	+ 0.5 cm.
Mean pressure	— 3.4

Valvular. Amount of air entering chest, 67.2 cc. per 1 k.

Average insp. pressure	— 8.6 cm.
Average exp. pressure	+ 15.6
Mean pressure	+ 35.

Open. The pressure oscillated about 1 cm. each side of 0.

Cardiograph records. Closed, rate increase = 17-20 per cent; excursion's decrease = 20-50 per cent.

Valvular; rate increase averaged 180 per cent, but increases as air entered even to 240 per cent, and excursion diminished.

Open; the excursion increased enormously, even five-fold, and the rate also increased to a point between the closed and the valvular. The differences between the two animals is due to their normal variations, the rabbit breathing fast and shallow, the dog slow and deep. In pneumothorax therefore compensation will be attained in the dog by faster, and the rabbits by deeper respiration. But dogs do not stand pneumothorax so well as rabbits and become more easily dyspnœic, since in the pleura is a greater negative pressure normally and greater displacements of organs is possible.

166. Unverricht. (A New Symptom for the Diagnosis of Lung Fistula in Pyopneumothorax. *Zeits. f. kl. Med.*, 1880, Bd. I, p. 536.

The patency or closure of the fistula is of the greatest importance in the treatment of pneumothorax, since this differs according as the fistula is open, closed, or valvular. The French and English believed open fistula was the rule. Skoda and other Germans thought it soon closed, but Skoda reckoned as closed cases where it was so by pressure, etc., and these are really valvular. Biermer, for the Germans, studied the question most carefully and took a middle ground. He sought for clinical symptoms to determine whether or not the fistula was still open.

Unverricht then discusses the various symptoms proposed; variations in amount of air, motility of the affected side, amphoric respiration, the "peculiar inspiratory sibilus" of the English which is pathognomonic in some cases, and gas analysis. He now adds the sign that on aspiration one hears a coarse, moist, gurgling sound at the end of inspiration with a

metallic ring, which he names Wasserpfeifengeräusch, and which is due to air bubbling from the fistula through the fluid when the chest is aspirated.

Two cases are reported in which this sign was of value. The conclusions are similar to those in his previous paper.

167. Delgrange. Observation de pneumothorax a la suite d'efforts. *Jour. des Sc. méd. de Lille*, 1880, II, p. 623

The patient was a youth, 18 years old, a coppersmith who had never been ill. As a result of an attempt to show the strength of his lungs he suddenly, while making violent expiratory efforts, developed pneumothorax of the left side. The dyspnœa was extreme. Recovery was rather slow.

168. Ross. A Case of Pneumohydro-thorax of Traumatic Origin. *Peoria Medical Monthly*, 1880-1, I, p. 192.

On the day before admission the man, a healthy person 35 yrs. old, was struck on the back by the pole of an omnibus. He was unconscious for one hour, and on regaining consciousness complained of oppression in the stomach and a lacerating pain in the right side of the chest accompanying a slight hacking cough. On admission respiration was 50 to the minute, the right side of the chest was tympanitic. On this side was movable dulness, amphoric respiration, metallic tinkling, and the succussion splash, and yet a slight bruise alone showed the external injury. In six weeks the chest was normal. Four years later he was reported to be well.

169. Riegel. Zur Diagnose des Pneumothorax. *Berliner klin. Wochensch.*, Vol. XVII, p. 709, 1880.

The author will discuss the diagnosis of the patency of the fistula. He recognizes the three forms, open, closed, and valvular. The idea that in open there is not the dilatation nor displacement of organs seen in the other two he considers false. Also in no case is that side of the chest quiet in respiration.

But in favor of an open fistula are:

1. Rapid changes in the amount of intrapleural air, as shown by the percussion tone and displacement of organs.
2. Change of pitch on percussion with the mouth open and closed. For this to be present the fistula must be large and open above the fluid.
3. "Maulvolle Expectoration," but a rare sign.
4. Demarquay and Ewald showed the importance of gas analysis.
5. Biermer showed the value of the percussion note after removal of some air. If the tone becomes again rapidly deeper the air is being renewed.

A *case* is then reported with autopsy, in which nearly every symptom of pneumothorax failed, but the bubbling sound heard as air entered the pleura through a fistula below the level of the fluid made the diagnosis positive. This was heard both with inspiration and expiration, air being

sucked in during inspiration and forced in during expiration, but later only during inspiration as the pressure in the cavity increased. Later doubtless that would also stop. Unverricht first called attention to this symptom but he produced it by aspirating air from the chest.

Describing this sound, he likens it to that produced by blowing air through a tube submerged in a vessel partly full of water.

The pleural walls were very much altered.

170. Coats and Inglis. Sudden Dyspnœa from Pneumothorax in Case of Comparative Good Health previously. Sudden Death. *Glasgow Med. Jour.*, XIV, p. 416, 1880.

The case reads like one of tuberculosis. Dyspnœa developed during hard work. Subcutaneous emphysema was present over the right mammary region which projected somewhat and on which side pneumothorax was present.

171. Biach. Zur Ætiologie des Pneumothorax. *Wien. med. Wochensch,*. Vol. XXX, 1880.

Biach, from literature and from the records of the three great Vienna hospitals, collected 918 cases of pneumothorax:

Phthisis	715
Pulmonary gangrene	65
Empyema	45
Trauma	32
Bronchiectasis	10
Emphysema	7
Abscess of the lung	10
Gangrene from infarction	4
Echinococcus of the lung	1
Ascaris lumbricoides in pleura	2
Following thoracentesis	3
Perforation of an encapsulated peritoneal exudate into the pleura	1
Perforation of a gastric ulcer	2
Perforation of an œsophageal ulcer	2
Perforation of a bronchial gland	1
Caries of the rib	1
Caries of the sternum	1
Fistulous communication from colon through liver to pleura from hydatid	1
Abscess of breast	1
Uncertain	14

Concerning the frequency of the condition in the Vienna hospitals he collected 433 cases, and in the same period these hospitals had treated 58,731 phthisical patients, hence the frequency was about 1 per cent.

Calculations of others are reviewed (Chambers 3.7 per cent; Lebert 5 per cent of fatal cases.)

Interesting cases are then reported at length or quoted from literature.

On page 63 he compiles the accident cases, 32 in number, and on page 142 is an interesting comparison of the frequency of pneumothorax following injuries as found in the records of one hospital during ten years.

These include crushing of thorax 227 cases, but not one with pneumothorax.

Penetrating wounds, 21. The result was pleurisy in 2, subcutaneous emphysema 2, pericarditis 1, not one case of pneumothorax.

Fractured clavicle 132 cases; 2 presented subcutaneous emphysema, none pneumothorax.

Fractured ribs 173; 14 cases presented subcutaneous emphysema, pneumonia 2, pleurisy 5, pneumothorax 2.

The article is a long one, the various sections beginning on pages 6, 37, 60, 142, 168, 406 and 431. The rare cases are reported or quoted.

172. Weil. *Archiv f. kl. Med.*, Vol. XXIX, p. 364, 1881.

Clinical Observations.

Forms of pneumothorax found in men.—Partial. Total.

Simple, with fluid exudate. Always at first is an open pneumothorax, later may be closed or valvular.

1. Closed. This means one in which no air enters or leaves the chest. It is "organically" closed when the fistula is obliterated or securely plugged. It is "mechanically" closed by pressure which presses the walls of the fistula together.

2. Open, air entering and leaving.

3. Valvular, in which case the air can enter during inspiration but cannot escape in expiration. After a certain amount of air has entered it is mechanically closed. Here belong most cases which originate in a pulmonary lesion. In open hydropneumothorax the fistula may be below the surface of the fluid, in which case the fluid is expectorated until the fistula is free again. To closed hydropneumothorax belong those cases in which the gas originates in the fluid.

In valvular hydropneumothorax the increase of fluid will raise the tension of the gas, and may mechanically close the fistula. If the fluid be aspirated the pressure is lowered and a mechanically closed fistula reopens.

Factors present in each group.—The retraction of the lung. Donders' work is quoted, who on the corpse decided the elastic tension to be 6 mm. Hg and added 1½ mm. for tonus, giving 7½ mm. Hg as the elasticity of the normal lung. But the retraction of a lung before death is much greater than that after death, as may be seen in animals, in the latter case the lung becoming much smaller and airless, even perfectly atelectatic after 1 to 2 hrs. of open pneumothorax.

Weil found the pleural volume of the same animal three or four times as great before death as post mortem, while Donders and Wintrich found the pressure at the trachea on opening the thorax only one-fourth or one-fifth as great after as before death. The retraction of the human lung

after death on opening the chest is more apparent than real, as may be seen if the chest be opened without cutting the ribs, also by measuring the air escaping by the trachea.

If the pleural volume of a man (the volume of air entering the chest to restore atmospheric pressure) be reckoned from the data of animal experiments it would be 1800 to 2400 cc.; this volume is allowed by the retraction of the lungs and the displacement of organs. That this is not a too great estimation is shown by the fact that a large amount of fluid may be present in the chest before the lung is collapsed, as measured by the negative pressure found before aspirating even 2000 cc. of fluid.

In the retraction of the lung there are two elements, elasticity and tonicity, which explain the extreme retraction during life if the chest be opened. This extreme retraction is important, since it closes the fistula, unless it be very large. Hence the variety of pneumothorax produced by a pulmonary wound depends on the form and size of the perforation, on the condition of the bronchus leading to the perforation and the capability of the lung to retract. The size of the hole is usually small, but a few millimeters in diameter. A great loss of substance is rare, since the pleura seldom tears over a large cavity, adhesions there protecting the pleural cavity. The perforation is usually of a small peripheral cavity. At first the air enters during inspiration and expiration, until the tension at expiration becomes the atmospheric. The perforation is smaller during expiration, hence after the expiratory pressure is positive less will escape than enters during inspiration, and the cavity will fill till no more can enter during inspiration. Hence what was at first an open pneumothorax is later valvular, and then mechanically closed. The closure is effected by the retraction of the lung, or its compression by the positive pressure of the gas, or the masses of fibrin at the orifice, or the collapse of the bronchus.

Open pneumothorax, in which the same amount leaves in expiration as enters in inspiration, is rare and can exist only when the fistula is large and has stiff walls. The more complete the retraction of the lung the better the chance that the fistula may be valvular. Hence an open pneumothorax is usually a partial one.

Differential Diagnosis of the Varieties of Pneumothorax.—Anatomical.—While absence of a fistula at autopsy points to closed pneumothorax, the discovery of one seldom indicates open, since it may intra vitam have been mechanically closed. The early diagnosis is important to determine the treatment, and the manometer gives the best data. The exudate may increase the tension or post mortem the cooling lower it. In Weil's 6 cases of tuberculous pneumothorax at autopsy the pressure was positive in all and varied from 3 to 14 cm. H_2O. The air which escaped varied from 50 to 560 cc. In some cases no opening could be found.

Clinically three forms can be distinguished. The loud metallic respiration similar to that heard in cases of external pneumothorax will indicate an open fistula, but the sign is not sure, since the absence of every respiratory murmur does not exclude an open fistula. Wintrich's change

of pitch, Laennec's metallic tinkling, Biermer's test, the dislocation of the organs, may all fail, and the gas analysis does not distinguish an organically closed from a mechanically closed fistula. Weil discusses Unverricht's Wasserpfeifengeräusch, the opinions of Riegel, Dance, and of others.

The rate of respiration is no aid, since a lengthened respiration is not seen in open cases. In valvular pneumothorax, however, the chest is at no time out of expiratory phase.

The manometrical method is the best and by following the change of pressure after aspiration one can separate organically closed from mechanically closed valvular cases.

173. West. Chronic Excavation of the Lung, Terminating in Localized Pneumothorax, with Extensive Opening into the Lung. *Trans. of the Path. Soc. of London*, 1881.

The patient was a phthisical youth 22 yrs. old, in whose case the diagnosis lay between pneumothorax and extensive cavity formation. "But such intense amphoric breathing and vocal resonance in pneumothorax could only be produced when the opening into the lung was very large, and the communication between the pneumothorax and bronchus direct," which was found at autopsy to be the case. Succussion and metallic sounds were not heard.

174. Cayley. A Case of Traumatic Pneumothorax from Swallowing a Fish Bone; Recovery. *Lancet*, 1881, II, p. 828.

The patient was a woman 48 yrs. old, who, eight or nine hours after swallowing a fish bone, was short of breath, and had the sensation of a load on the chest. Left-sided pneumothorax was found. She was discharged well on the sixteenth day. The writer thinks the bone pierced the lung rather than the œsophagus. There was no hæmoptysis.

175. Auquier. Du rôle des tensions et des pressures intrathoraciques dans le pneumothorax traumatique. *Gaz. hebdom. des Science. méd. de Montpellier*, Nov. 18, 1882.

Page 557. The text of this article is the case of a man badly injured by being run over by a cart. The diagnosis was "Fracture of several ribs and the sternum, dislocation of the shoulder, tear of the right lung, right hæmopneumothorax, subcutaneous emphysema of the left side. Reduction of the dislocation interrupted by threatened syncope. Increased intrathoracic pressure on the right pleura due to the pneumothorax. Threatened asphyxia. Thoracentesis, immediate relief. Spontaneous reduction of the scapulo-humeral dislocation. Recovery.

The chief interest in this long paper lies in the number of references given to the literature of pneumothorax which are seldom mentioned elsewhere.

Page 598. The writer discusses fracture of the ribs and especially the absence of suppuration of those compound fractures which open into the lung. The explanation given for this absence is the lack of oxygen neces-

sary for the growth of the bacteria. He then mentions the rare coincidence in this case of pneumothorax without external emphysema of the right side and external emphysema without pneumothorax on the left.

Page 609. The article now becomes more interesting. He says it was Van Swieten who first said that the intrapleural vacuum holds the lungs against the chest walls, thus counterbalancing their elasticity. Bérart again taught this theory, and Richet developed it further, i. e. the antagonism between the elasticity of the lung and the intrabronchial tension. But Auquier disagrees: "Traumatic pneumothorax is extremely rare" (Richet). And experimental evidence against the inherent tendency to the formation of pneumothorax is found in the work of Dolbeau and Smith (1868), who likened the cohesion of the pleuræ to that of two plates of wet glass. Duplay (*Traité de path. ext.*) explicitly states the presence of a physical adhesion between the pleuræ which overcomes the elasticity of the lung. The writer urges the likeness of the pleuræ to the plates of wet glass, and insists that a cannula in the pleural cavity is not in a normal chest but in a small pneumothorax cavity. Clinical and experimental evidence force him to the conclusion "that the tendency to an intrapleural vacuum does not exist," and that the pleuræ are held together not by the atmospheric pressure but by a molecular force greater than the elasticity of the lung.

Page 617. The reason why a traumatic pneumothorax should arise is now discussed and his conclusion is that the injury must separate the pleuræ at a point and allow a certain amount of air to enter. Whether or not the pneumothorax will become general, depends on whether the amount of lung liberated from the costal pleura is sufficient that its elasticity may overcome the cohesion at the periphery of the local pneumothorax. Smith pointed out that in the distension of a collapsed lung the sound lung takes an active part, but Auquier calls his attention to the previous work of Morel-Lavellée (1850), whose statements to the same effect he quotes. Beaunis showed that in forced expiration the intrapulmonary pressure could be + 87 mm. Hg., hence the expansion is easily explained, expiration being the active factor and the molecular adhesion holding the gain already made. Smith thought that a perforation to cause pneumothorax should be not in the center of a lobe but at a border. Auquier elaborates this view, calling attention to the two forces at right angles to each other, of which that causing the retraction of the lung to its hilus may be considered the resultant. The one is at right angles to the ribs and exerted at the middle of a lobe's surface, the other at the border of the lung is at right angles to this and causes the gliding motion of respiration. The resistance to retraction offered by the molecular cohesion diminishes progressively from the center of the surface to the edge. He considers a rational classification of pneumothorax to be: lateral (in which the air is near the middle of a lobe, the margins of which do not become separated from the chest wall), intralobular, monolobular, bilobular, and total.

176. Stephanides. Ein seltener Fall von Pneumothorax. *Wiener med. Wochensch.*, Vol. XXXII, page 996, 1882.

The *case* is considered one of pneumothorax resulting from a gastro-duodenal ulcer, interesting because the pneumothorax was on the right side. (In Barlow's and in Sturges' cases it was left-sided.) In this case also there was only gas in the pleura, hence the recovery was rapid.

177. Vetlesen. (A Case of Diffuse Persistent Pneumothorax.) *Centralbl. f. klin. Med.*, Sept. 23, 1882.

The *case* was a young woman, 18 yrs. old, with pneumothorax following the rupture of "a small tuberculous superficial cavity." No effusion, no fever followed. Of interest was its chronicity, for it continued about seven months, the patient feeling quite well. Of interest also was a dry murmur in the trachea accompanying the end of inspiration and the end of expiration, which the author considered to be produced by the vibration of a valve of the fistula.

178. Orlebar. Some Cases of Pneumothorax: their Result and Treatment, together with a Description of a Cannula with a Valvular Attachment. *Brit. Med. Jour.*, Nov. 18, 1882.

Case 1.—Hydropneumothorax.

Cases 2-6.—Evidently tuberculous.

In case 5 a valvular cannula was kept in place for several weeks. The patient is improving.

179. Natorp. Doppelseitiger Pneumothorax in folge vicariirenden Emphysems. Würzburg, 1882.

This thesis was written under Prof. Gerhardt's direction. The case was one with pure pneumothorax on the left side, but the right lung was the seat of chronic tuberculosis and an encapsulated pneumothorax. The right lung was almost excluded from respiration by disease; the left lung, itself diseased, was unable to compensate by vicarious emphysema, and pneumothorax resulted.

The writer reports from literature seven cases of double pneumothorax, and from the Würzburg clinic 38 cases of pneumothorax. These are briefly stated.

180. Weil. *Deutsch. Arch. f. klin. Med.*, May 30, 1882.

B. Clinical Section.—Analysis of fifty-five cases of pneumothorax. These cases are those in the records of Friedrich's clinic during the past seventeen years. Of the fifty-five, forty-six were due to phthisis, six to empyema, and three doubtful. Weil now quotes the compilations of cases already reported.

1. *Cases due to Empyema.* The six cases are reported at length.

2. *Cases of doubtful etiology.* These three cases are also reported at length.

3. *Tuberculous Cases.—Etiology—Frequency.* These cases were from a total number of 1200 tuberculous cases treated, giving a frequency of 3.8 per cent. But he considers this figure valueless, and considers only

the cases which came to autopsy; of these there were 36 pneumothorax cases in 355 cases of tuberculosis, i. e. 10.1 per cent. He considers this figure nearer the truth. The reason for excluding the other cases is that the future history of a consumptive who left the hospital could not be followed and some must later have developed pneumothorax.

The *sex* (35 of the 46 were men) and the *age* are discussed; 14 were right-sided, 31 left-sided and 1 bilateral. There were recurrences on the same side in 3 cases. The reason for the predominance of the left-sided cases is discussed. (In all these points the literature is well reviewed.)

In answer to the question whether pneumothorax developed on the *side with the most advanced tuberculosis* he found that in 74.3 per cent of the cases that was true. He considers that pneumothorax develops more commonly in acute cases or in chronic cases with an acute exacerbation. None of the cases were afebrile. It might develop in long standing or early cases.

The immediate cause is now discussed, the *anatomical findings, position of the fistula*, etc. In 20 of the 36 autopsy cases the pressure was positive. The rule is for the fistula to remain open for weeks or months, although it may become organically closed in a few days. The most motile part of the lungs, both from respiratory as from cardio-pneumonic movements, is the anterior edge of the left lung at the incisura cardiaca, and this may in part explain the predominance of perforations on the left side.

The *pleurisy* is now discussed. In one case after nine weeks' duration there was no exudate, but the great majority, except the rapidly fatal, developed a pleurisy which in the majority of cases was purulent. He explains (differing from Senator) the common absence of a purulent exudate and decomposition on the ground that the perforation is so soon mechanically closed, and doubts that any really open cases are without a purulent exudate.

Pressure Symptoms.—In all but two cases, and in these thoracotomy had been performed, pressure symptoms were noted. Distension of the affected side, widening and tension of the intercostal spaces which projected outward, low position of the diaphragm, were found with great regularity, even in the partial cases. The dislocation of the heart was marked, but Weil thinks that only in the case of moderate dislocation does the heart move in toto, and that usually the apex becomes slightly raised, since the heart rotates somewhat about its base as a fixed point.

Symptomatology.—Onset sudden, as a rule, with dyspnœa, pain, rapid pulse, and initial drop of temperature. Cyanosis and collapse were present in some cases. An interesting early symptom was œdema of the hand of the affected side.

Physical Signs.—Inspection; percussion; the most important signs being hyperresonance over a wider area than normal. Auscultation gives as most important sign silence over the affected side. This is the rule in an early case over the anterior chest wall, and in only about one-half the cases could amphoric breathing be heard in the side and back. For the

metallic phenomena the chest cavity must have good amphoric qualities. First the shape must be regular and the walls smooth. Second, to transmit the sound the wall of the chest must have a certain thickness and tension; and thirdly, the sound to be propagated must have a certain pitch. All these conditions are present as a rule in pneumothorax, and the metallic phenomena can as a rule be elicited by coughing, speaking, percussion, etc., if not by breathing. (Plessimeterstäbchen-percussion he ascribes to Huebner and says Leichtenstern developed it.) Yet in some fresh cases no such phenomena can be obtained; perhaps the tension of the wall is too great, since, as Traube taught, percussion of the dead body may give a metallic note when the same was absent in life, or it may appear first after puncture of the thorax (Fräntzel). In some cases the architecture may not be adapted for its production, but if this element is important it is surprising it so often occurs. The trouble may lie in the pitch and intensity of the sounds themselves. He considers the murmur produced by the movement of air through a truly open pneumothorax fistula as best adapted. In other cases it is the sound in the larynx, trachea, bronchi or neighboring cavities which acquires the metallic quality, and the fact that the last are important is shown by the exquisite metallic sounds produced in a case where extensive adhesions have prevented the collapse of the lung. But harder to explain is the localization of the amphoric tone, for this is usually heard only at the lower back. Perhaps the acoustic properties of the chest explain this, but certainly it is not the proximity of the fistula. Other metallic phenomena are the metallic tinkling, voice, cough, succussion splash, and in but two cases of left pneumothorax, heart tones. Leichtenstern explained the rarity of the metallic quality of the heart sounds by their lower pitch.

The Biermer's change of pitch on percussion by opening and closing the mouth, Weil could not verify. Certain modifications appear when an exudate forms. The succussion splash is heard, and dulness is not heard, since the fluid occupies the concavity of the displaced diaphragm. Considerable fluid may not be detected in the chest, e. g. Senator's case, which was hyperresonant to the twelfth rib after introducing 800 cc. of dilute salicylic acid into the pleura in addition to the fluid already present. If enough be present to give a line of dulness, in no other condition is movable dulness so marked.

The febrile features are next discussed.

181. Weil. *Deut. Arch. f. klin. Med.*, Vol. 31, page 243, July 13, 1882.

Analysis of 55 cases (continued).

Duration — Course — Termination.—In one-half the cases death occurred in the first month, in one-fourth in the first week. Reasons for this difference in duration are given. In four cases the air disappeared and was replaced by fluid, in one case the cure was complete. Five cases with hydro- or pyo-pneumothorax which remained in good condition for some time (even 2¾ yrs.) after leaving the hospital, are reported at length.

Weil discusses the cases of phthisis which were not unfavorably influenced, but even improved by the pneumothorax.

Therapy.—A chapter of twenty-six pages is devoted to the treatment by operation. We will give only Weil's conclusions:

1. The majority of cases of tuberculous pneumothorax present a bad outlook for operation. In these hopeless cases operation is undertaken only as a last resort.

2. In the minority of cases, those with a relatively good prognosis, operation may result in improvement or even recovery.

3. In the first five or six weeks from the onset operation should be performed only in case of threatened suffocation, since the fistula is not usually then healed.

4. If soon after the onset the dyspnœa is extreme and ice bags and morphine fail, puncture may be resorted to. If the pressure again rises a permanent cannula or incision will alone keep the pressure down.

5. Should operation be necessary in the first few weeks the fluid should be aspirated, since this is usually the factor at fault. If this fails, incision alone is of value.

6. In favorable cases operation is justified when there is no severe indication, but not until after the fourth to the sixth week, for then the fistula may be considered organically closed, and the patient will probably be without fever.

7. If in such a case the fluid is compressing the air, the fluid should be aspirated, but only moderate amounts at a time.

8. In the case of sero-fibrinous exudates small quantities (500-1000 cc. at a time) may be removed.

9. If the fluid is purulent Senator's procedure may be used.

10. If the pus quickly re-collects (and the case favorable), incise at once.

11. If at the end of five or six weeks there is still much gas but little fluid, Weil recommends aspiration of air.

12. Aspiration of both air and gas is advised for a stationary case with a fair amount of fluid and considerable gas.

Bard in discussing Weil's work says, that he made two errors.

First, in closed pneumothorax he neglected the rapid absorption of air.

Second, working with pneumothorax opening externally to the constant atmospheric pressure, he neglects the fact that in the case of pneumothorax from perforation of the lung the fistula opens into a cavity (the bronchial tree), the pressure of which is neither atmospheric nor constant. Weil should not apply his animal experiments to clinical cases.

182. Johnson (G.). Pneumothorax. *Lancet*, Mar. 4, 1882.

This case of acute perforative pneumothorax, with perfect recovery,

occurred in a boy 15 yrs. old, who ran in a "paper chase" about twenty miles, and then fell down exhausted. The following two days he was well, but on the third, after running up stairs, he was seized with pain in the left side, urgent dyspnœa and great prostration. On the next day all of the signs of pneumothorax were found present. Eight days later he felt well. The left side of the chest was nearly motionless and hyper-resonant everywhere except in the interscapular region, where it was slightly duller than the corresponding point of the other side, and here a faint respiratory murmur was heard. Elsewhere over the whole left side was distinct amphoric blowing, with occasional metallic tinkling and amphoric echo of the voice and cough. There was no dulness at the base nor succussion splash. The heart was to the right of the sternum. Thirteen days later there was some respiratory movement on the left side, the amphoric blowing and metallic tinkling had ceased, there was some respiratory murmur on the left side, and the heart's impulse had returned to that side.

Twenty-six days later the boy was practically well.

The following explanation was suggested: Two years before he had had a chronic cough. The apex of the left lung may at this time have become adherent, and the violent exertion of running have torn these adhesions and the pleura.

In the discussion of the case **Dr. Douglas Powell** discussed the loud amphoric breath sound: whether this was due to a modification of the normal breath sound by a large air cavity, or to the to and fro rush of air into the pleura. He had recently seen a case of a greatly distended stomach over the area of which the most typical amphoric breath sound was heard. . . . In some cases where the pneumothorax occurred on the more diseased side it has seemed to retard the lung mischief and to prolong life.

Dr. F. Taylor mentioned a case he had had of hydropneumothorax the sequel of an acute pneumonia, an hitherto unrecognized occurrence.

Dr. Pearce Gould mentioned a case of a child who had been run over. Pure pneumothorax followed. There was no broken rib. Respirations, 60 to the minute. The pleura was tapped, air escaped with a loud hissing noise. In one week the chest was again normal.

Mr. H. Marsh mentioned a case of a man with left-sided pneumothorax following a run-over accident. The chest was twice tapped and air escaped, but reaccumulated. He died. At autopsy the lung was found torn quite away from the left bronchus. Two other cases of rupture of the lung without fracture of the ribs (one at least in an adult) were mentioned.

A case following perhaps pyæmic abscess, and two other spontaneous cases are mentioned.

Mr. Lister said one case mentioned reminded him of those cases of fractured ribs, in which, if the wound in the costal pleura healed, grave or even fatal pneumothorax occurs. "If a piece of lung be taken and a glass

tube tied into a bronchus and that connected with a syringe full of air, and if a free incision be then made into the lung substance and the piston (of the) syringe depressed, the air passes into the wound of the lung very readily. But if now the attempt to raise the piston be made it is found to be impossible to do it without great force, and if the force be removed the piston flies back again. This shows that a wound of a lung is a valved opening letting air pass out but not in. In the case reported above as the amphoric sound occurred in both inspiration and expiration it must (it cannot) have been caused by the rush of air through the opening. [The parentheses are ours, enclosing proposed emendations to the text, which seems at fault at these points.]

Dr. G. Johnson thought the amphoric respiration due to the passage of air into a large hollow cavity, for in an empyema case with external opening it was present, but stopped when the finger closed the hole.

183. Gould. Rupture of the Lung; Pneumothorax; Paracentesis; Remarks. *Lancet*, Jan. 14, 1882.

The child, aged six years, was run over by a cab, two wheels passing over the belly. Left-sided pneumothorax followed. No ribs were broken. Two days later he was very dyspnœic, respirations 60 to 70 to the minute. The left intercostal spaces were wide, the walls were fixed, the percussion note tympanitic. The respiratory sounds were amphoric, with very distinct metallic tinkle and bell sound. Apex beat at the xyphoid cartilage. A trocar was inserted, air escaped with a hiss, and then with each expiration (its return was prevented). This was continued until blood escaped. The relief was marked and instantaneous; the boy lay on his back, respirations 48 to the minute, and the apex beat in the normal situation. The physical signs of pneumothorax had all disappeared. The hæmothorax soon cleared up.

In this case the air was apparently completely evacuated by the one tapping.

184. Boursot. Contribution à l'étude des signes du pneumothorax. Paris, 1883.

The conclusions of the author of this thesis are that one of the most important signs of pneumothorax is given by the combined percussion and auscultation of the anterior region of the chest. It was the presence of a more or less marked hyperresonance contrasted with the complete absence of the respiratory murmur in the same zone, which in an uncertain case placed the diagnosis beyond doubt. From it may be determined the extent of the pneumothorax whether circumscribed or general.

Ten cases are reviewed.

185. Seifert. Pneumothorax. *Deutsch. Arch. f. klin. Med.*, Vol. 33, p. 157, 1882.

After stating Weil's work and his method of diagnosis of the form of pneumothorax from the manometrical pressure determination, Seifert

reports at length two cases of tuberculous pneumothorax in which the diagnosis of the open form could be made on the dead body, the atmospheric pressure obtaining in each case. In Case III the positive pressure of 3½ cm. found at autopsy indicated a closed pneumothorax which was found to be the case, a closed pneumothorax following a valvular. In Case IV the measurements were made during life, expiratory pressure + 6.5 cm. H_2O, inspiration + 5 cm., hence the closed form was diagnosed. On the dead body the pressure was + 3.5 cm. These manometrical observations and gas analyses to determine whether the closure be organic or not, he thinks will settle the diagnosis of the form of the fistula.

186. Clark (F. le G.). Emphysema and Pneumothorax. *St. Thomas Hosp. Rep.*, Vol. XIII, page 9, 1883.

Clark, without specifically reporting cases, speaks of the comparative infrequence of injury of the lung following fractured rib, and explains the mechanics of it by the fact that as a rule ribs are fractured by overbending, hence the fractured ends do not encroach upon the pleural cavity. But fractures due to direct violence, however, do wound the lung and either emphysema or pneumothorax may result; yet if uncomplicated neither need be fatal.

187. Bähr. A Statistical Contribution to Pneumothorax. Würzburg, 1883.

The writer reviews the forty cases of pneumothorax occurring in Gerhardt's clinic at Würzburg, during the years 1873-81. No surgical cases are included.

Of these 40, tuberculosis caused 36; empyema 3, and pulmonary abscess 1.

Contrary to Weil's experience, of the tuberculous cases 20 were of the right and 16 the left side.

The pneumothorax was in 26 cases total; in 9 circumscribed. In 2 cases no fluid was present.

The minute analysis of the cases follows.

188. West. Complete Recovery from Idiopathic Pneumothorax Without Effusion. *Brit. Med. Jour.*, Dec. 22, 1883.

A man, 24 years old, tuberculous with pneumothorax, was relieved by aspiration, and in six weeks was well. (The discussion was interesting.)

Mahomed had seen cases of typhoid in which the condition of the lung was such as to render the production of pneumothorax imminent. The lung was in a peculiar spleen-like state, which follows prolonged fevers.

Jonathan Hutchinson had "seen many cases of pneumothorax following fractures of the ribs."

Page had seen pneumothorax follow fracture of the first rib.

Pearce had seen a case of ruptured lung result from a run-over accident without fractured ribs.

Maclagan mentioned the case of a gentleman thrown from his horse against a tree. Pneumothorax resulted, but no ribs had been fractured.

Taylor had had a case of pneumothorax after acute pneumonia. (This led to a discussion concerning the tympanitic tone heard over a pneumonic area.)

West in conclusion asked for data concerning pneumothorax following fractured ribs. He considered it rare. He considered the displacement of organs one of the best signs of pneumothorax.

189. v. Ziemssen. Pneumopyothorax. *Ein Sommersemester in der Klinik des Herrn Prof. v. Ziemssen* (1883), p. 20.

This is a clinical lecture. Ziemssen says he had punctured the chest repeatedly with a capillary trocar to relieve the intrapleural pressure and the dyspnœa, and had repeated it many times with favorable result. The subcutaneous emphysema following such a procedure is due to coughing which can be prevented by morphia. He attributes the distension of the chest above the inspiratory position in good part to the warming of the air introduced cold.

The gas in this case was analyzed:

CO_2	11.32 per cent
O	8.15
N	80.53

190. Biach. Über Heilung des Pneumothorax. *Mitth. des Vereines der Aerzte in Nieder-Oesterreich*, Vol. IX, p. 282, 1883.

The writer reports a case with recovery and collects from literature 78 cases of recovery. In 68 cases the cause of the condition was stated.

Trauma	19
Phthisis	11
Unknown cause	24
Idiopathic	3
Metastasis	1
Bronchopneumonia	1
Chronic pneumonia	4
Lung abscess after pneumonia	1
Pleurisy	1
Emphysema	3

191. Coupland. A Clinical Lecture on Pneumothorax. *The Clinical Journal*, London, II, p. 225, 1883.

This lecture states Coupland's experience with this disease. An interesting case is reported at length, in which the markedly amphoric quality of breath sounds and of the vocal resonance, was perhaps due to the comparatively large size of the fistulous opening, and its free communication with the pleura.

192. Lyman. Pneumothorax. *Boston Med. and Surg. Journal*, Dec. 18, 1884.

Lyman presented three cases of pneumothorax before the Boston Society for Medical Improvement. One was of surgical, one of tubercular origin, and one followed pleurisy.

193. West. A Contribution to the Pathology of Pneumothorax. *Lancet*, May 3, 1884.

"The material for this paper was obtained from the records of the City of London Hospital for Diseases of the Chest, Victoria Park, extending over a period of twenty-eight years, from 1856 to 1883, inclusive."

One hundred and one cases are tabulated, and all but two were clearly due to phthisis, and these two may have been, although emphysema is assigned as the cause. The mortality was 65.4 per cent. He calculates that 5 per cent of all tuberculous cases die of pneumothorax. He mentions Powell's, Lebert's, similar figures, Weil's 13 per cent, Chambers' 3.7 per cent, and Biach's 1 per cent.

He discusses sex; 23 were women and 75 men; the sex mortality was 56 per cent for the men, 87 per cent for the women; side affected; age, over half were from 25 to 35 years old; duration, 75 per cent dying within the first fortnight, and 90 per cent before the end of the first month; effusion, which is common but not necessary, the chances of its being purulent increasing with the duration of the case; the perforation, usually single, found in two-thirds of the cases in the upper lobe.

The reader is referred to the original article for more detailed summary of the cases. The above is very much abbreviated, and the figures confirm for the most part those given by previous writers.

194. Desmaroux. Idiopathic Pneumothorax. *Gazette des Hôpitaux*, Vol. 57, p. 804, 1884.

A *case* is reported of a healthy man, 25 years old, who suddenly was seized with repeated chills and extreme pain in the right side; there was no cough, no traumatism preceding. Pneumothorax was found on examination. Since a tear of the lungs would mean hæmothorax with the signs of effusion, he says "making certain reservations it is permitted to admit with Laennec and Professors Graves and Stokes that the . . . . . pleura has the ability to exhale an aeriform fluid and to give rise . . . . . to an idiopathic pneumothorax."

195. Hayden. Pneumothorax. *The Cincinnati Lancet and Clinic*, Vol. XIII, p. 98, 1884.

The mechanics of the disease is first discussed. The case, a colored man, 48 years old, had tuberculosis, and in about three months pneumothorax of the left side. The chest was aspirated and much gas withdrawn but no pus. One week later he expectorated large amounts of pus; two weeks later he died.

Autopsy.—The left pleural cavity was distended with gas, which escaped with considerable force. The left lung was almost completely destroyed and gangrenous. The pleura contained about a pint of pus. In a cavity in the lung was a pint and a half of pus. The right lung was tuberculous. (The gas was removed by suction with an aspirator.)

196. Cayley. Pneumothorax Occurring During the Course of Typhoid Fever. *Trans. of the Clinical Soc. of London*, Vol. XVII, p. 52, 1884.

The patient was a girl 14 yrs. old with a severe attack of typhoid fever. On the 15th day of the disease bronchitic symptoms were present, and six days later a right-sided pleurisy. On the twenty-third day "the patient was much worse, the temperature at night was 104° . . . the breathing embarrassed. There was severe pain and tenderness over the right side of the chest and now a friction sound was audible, mixed with abundant fine crepitant râles, but there was no dulness." Two days later she had a severe attack of dyspnœa lasting about fifteen minutes, accompanied by symptoms of collapse; "this attack I presume coincided with the rupture of the lung." The chest remained resonant, but the respiratory movements, voice and breath sounds became very feeble (her condition made minute examination impossible). Two days later were noted distinct tympanitic resonance with amphoric breathing over the right chest. On the 28th day was another very severe attack of dyspnœa lasting fifteen minutes with extreme collapse, but the patient rallied. The following day she was better. The right side of the chest was bulging and tympanitic; amphoric echo, metallic tinkling, and an exquisite coin sound were everywhere present. The 30th day of the fever another slight attack of dyspnœa. The patient improved steadily. On the 37th day the orthopnœa continued and the heart was displaced to the left and down. To relieve the tension a fine trocar was inserted, air hissed out. This gave considerable relief to the dyspnœa, but produced no change in the physical signs. This was repeated two days later and a few drops of pus escaped with the air. During the fifth week after the onset of the pneumothorax the signs of it had disappeared. The patient was up and about. The duration of the fever seems to have been about 26 days.

"Although not a common complication of typhoid fever, many cases of pneumothorax have been recorded. Sometimes it is caused by pulmonary gangrene. Dr. Gardiner has published a case where small subpleural collections of pus were found on post-mortem examination. The present case must in all probabilities be ascribed to the breaking down of a pulmonary embolism due to thrombosis of the right cavities of the heart from asystole."

197. West. Case of Complete Recovery from Pneumothorax without Effusion of Fluid. *Trans. of the Clinical Soc. of London*, Vol. XVII, p. 56, 1884

The patient was a man 24 yrs. old, who had for some weeks been con-

sidered phthisical, though there were no distinct physical signs of this disease. One evening (Nov. 7) he was seized wih severe pains in the right side, and dyspnœa, and presented the signs of right pneumothorax. The next day a large amount of air was drawn off through an aspirating needle. Four days later the "respirations were 28. The right side was tympanitic to percussion in every part, the resonance extending to the eighth rib on the nipple line and to the twelfth posteriorly, and for some distance to the left of the sternum, the heart being displaced and its apex beat one inch outside the left nipple line. There was no distension of the side to sight or touch. A loud bell sound was audible from back to front, and amphoric breathing was well marked over the whole side, but succussion was absent." There was considerable subcutaneous emphysema over the whole right side, due probably to the paracentesis.

Nov. 29. The physical signs were unaltered except that the apex of the heart had returned to the nipple line.

Dec. 20. The right side seemed a little flatter and to move somewhat less than the left, but amphoric sounds and the bell sound were absent, and natural breath sounds were audible over the entire side. The apex of the heart was half an inch inside the nipple. Patient discharged.

West considers the deficiency of oxygen in the gas removed (only qualitative tests were done) proof that the fistula was closed. "The marked amphoric breathing is . . . so far as my own observation goes, almost always present when there is a large patent opening, still it is also frequently observed when the opening is certainly closed. What the cause of amphoric breathing is in such cases has not been, I think, satisfactorily explained. It is, however, interesting to observe that this physical sign gradually disappeared as recovery progressed." He considered the cause phthisis and thinks the effusion of air checked the disease. West found in literature twenty-four similar cases (with recovery). Some were well in a few days, others in a few weeks, the longest took eleven weeks (Schrötter's case, *Wochensch. d. Gesell. d. Aerzte*, 1876). In 18 cases the probable cause was specified:

4 certainly phthisis.
4 probably phthisis.
5 emphysema.
2 Overexertion of strong, healthy persons.
3 Traumatic.

A table of the cases is given.

198. Roser. Der offene Pneumothorax. *Deut. med. Wochensch.*, Feb. 19, 1885.

Roser for fourteen years produced purposely on his empyema cases a pneumothorax and during the last twenty years resected ribs in all cases that the opening into the pleura might be a larger one. With a mirror one can directly inspect the condition of the pleural cavity. He recommends to produce open pneumothorax in certain old empyema cases in which the extensive resection of the ribs necessary to obliterate the pleural cavity would be a dangerous procedure.

199. Cornils. Ein Fall von Pneumothorax mit par distance Hörbaren Herztönen. *Deut. med. Wochensch.*, June 11, 1885.

The case was a young man 23 yrs. old in the last stages of consumption, whose symptoms suddenly became worse. No diagnosis was made of the cause. At times three persons heard the heart beat at a little distance from the patient, but the phenomenon lasted only a few moments and could not at will be made to return. At autopsy a partial pneumothorax occupying the space of the left lower lobe was found without fluid and with smooth walls.

200. West. Cases of Pneumothorax. *Trans. of the Clin. Soc. of London*, Vol. XIX, p. 227, 1886.

West adds to his previous report (Vol. XVII) three more cases.

Case 1.—Pneumothorax, right side, in the course of severe phthisis; death after two days, chiefly due to suffocation by the contents of a large cavity forced out into bronchi by compression.

Case 2.—Pneumothorax, right side, three weeks duration on admission . . . profuse hæmoptysis, tapped twice and subsequently free incision; death from exhaustion; . . . aneurism of pulmonary artery. Before the second tapping the intrapleural pressure was $+2\frac{1}{4}$ in. H_2O on expiration, and $+ \frac{1}{2}$ in. on inspiration.

Case 3.—Pneumothorax right side . . . paracentesis twice; complete recovery; patient well and hard at work three weeks afterwards.

At the first aspiration the maximum expiratory pressure was 33.5 cm. H_2O; minimum inspiratory pressure, 17.25 cm. H_2O; mean pressure, 25 cm. H_2O. Analysis of the gas showed 12 per cent CO_2. Three weeks later at aspiration the maximum pressure was 8 in. of water.

201. Weil. Weitere Mittheilungen über Pneumothorax. *Deuts. Arch. f. klin. Med.*, Vol. 40, Nov., 1886.

Weil reports his experience since his last communication (a little over two years). After briefly reviewing his former work he reports seven cases of tuberculous pneumothorax (6 men and 1 woman) among eighty-four phthisical cases. In all cases that pleura was involved on the side where the tuberculosis of the lung was most advanced. All were cases of acute or subacute phthisis, and he is confident that acute phthisis is a much more favorable ground for pneumothorax than chronic. He never saw pneumothorax develop in a non-febrile case of tuberculosis. The cases developed early in the course of the disease.

In eleven cases the intrapleural pressure had been determined. In all tuberculous cases the pressure was found positive, from 2 to 10 mm. Hg. The amount of air which escaped was from 10 to 720 cc. Hence all were valvular cases. The perforation in his experience remains open for weeks or months. Other anatomical features are discussed.

The metallic phenomena, especially the amphoric respiration, are deceptive, since they failed in the fresh cases, especially over the front, except perhaps at the apex, where the lung is adherent. They are heard espe-

cially at the lower posterior and lateral parts of the chest. Their point of maximum intensity was in no case the position of the perforation. They were not, therefore, in these cases caused by the air blowing in and out of the perforation. He considers them due to murmurs propagated from the larynx, neighboring bronchi and cavities. Yet the absence of a murmur near the perforation means its closure.

Granting these metallic phenomena are not of themselves evidence in favor of open pneumothorax, since they are present where the fistula is closed; granting on the other hand that failure of all such murmurs means closed pneumothorax, he believes that there is a blowing murmur like that heard in externally open pneumothorax which does mean a fistula open internally. Hippocratic succussion splash and movable dulness were as a rule present not before the third day, unless a pleurisy had preceded. The exudate usually increased progressively; even a pyopneumothorax case could be non-febrile.

Six cases were closed valvular pneumothorax, one an open. He describes the mechanism of a valvular fistula, considering the air to enter during inspiration till no more can, when we have a mechanically closed valvular pneumothorax. The symptoms of this are positive pressure of the gas, great compression of the lung, the absence of an amphoric blowing murmur and the pressure symptoms.

To show the value of manometrical determinations in the diagnosis of the fistula he reports a case in which at first the pressure on inspiration of — 2 mm Hg., expiration + 6 to 8 mm. Hg., average + 3 mm., indicated a mechanically closed valvular fistula. One month later, the dyspnœa being marked and the amount of exudate having increased, the chest was again aspirated.

Insp. — 2 mm. Hg.; expir. + 30 mm.; average + 14 mm.

After 200 cm. of air were removed, insp. 0 mm.; expir. + 10 mm.; average + 5 mm.

After 400 cm. of air removed, insp. — 1 to — 2 mm.; exp. + 6 mm.; average + 2 mm.

After 600 cm. of air removed, insp. — 5 mm.; exp. + 3 to 4 mm.; average — 1 mm.

After coughing the pressure remained the same, hence an organically closed fistula was diagnosed.

1200 cc. of pus were now removed, after which the pressure on insp. was — 8 mm.; exp. + 3 mm.; average — 2½ mm.

Twelve days later the same operation was repeated.

	Insp.	Exp.	Average.
At first	+ 0	+15 mm. Hg.	+7.5 mm.
After 100 cc. air removed.	— 2	+10	+4
After 200 cc. air removed.	— 5	+ 8	+1.5
After 400 cc. air removed.	— 7	+ 6	—0.5
After 600 cc. air removed.	—11	+ 2	—4.5

Then 600 cc. of pus were removed.

Autopsy showed no fistula.

Open pneumothorax (the air entering and leaving easily) is rare and possible only when the lung cannot retract, hence when many adhesions are present. Such a case will usually be a partial pneumothorax. He cites such a case.

The duration, course and termination are now considered.

Pneumothorax due to pulmonary gangrene; recovery. Two such cases are reported at length; partial pneumothorax in each case, healing in three weeks.

202. Adams. A Case of Pneumothorax. *Boston Med. and Surg. Jour.*, Vol. CXV, p. 397, 1886.

Adams reports the case of a man 46 yrs. old, with left-sided pneumothorax due to straining at stool. The signs of this condition lasted about four years. Then he was considered well.

There was no effusion at any time, no movable dulness, no metallic tinkling

Adams quotes other cases of long standing.

203. Gimbert. Pneumothorax, vergetures de la peau du côté opposé. *La France médicale*, July 3, 1886, II, page 905.

The patient, a young man 17 yrs. old, had had hæmoptysis the preceding August, followed by cough and the signs of tuberculous bronchitis. On examination in July, 1886, both lungs were involved, the general condition of the patient was poor, and the outlook bad. In December was found caseous pneumonia of the right upper lobe, which later involved the right lung. In January there was present in the right back a circumscribed pneumothorax. The chest became much distended. Intercostal neuralgia developed on the left side, and the skin was there found to present three groups of weals, the largest 1 cm. broad and 8 to 10 cm. long. Their arrangement is described.

The writer discusses their cause. He discusses Thoan's case, who considered them due to trophic disturbances, but Gimbert believes in both cases the mechanical distension is sufficient and the good side is the seat of the weals, since in the coughing and other respiratory movements this is the side most concerned.

204a. Bunge. How is the Absorption of Air from the Pleural Cavity to be Explained? *Archiv f. Physiol.*, Suppl. Bd., 1886.

The absorption of air from the pleural cavity after closure of the fistula has never been explained. Bunge proposes to do so, using the conclusions of Ehrlich (*Das Sauerstoffbedürfniss des Organismus*, Berlin, 1885), who by the use of blue dyes, which by reduction were decolorized, found that no tissue had so much power to remove O as the surface of the lungs. Hence all O is soon removed from the pleural cavity by reduction processes. As soon as the O is consumed the partial pressure of the N nearly equal to that of the atmosphere is higher than that of the surrounding tissues in which it can rise little over four-fifths of an atmosphere, hence is absorbed by the blood and slowly diffused from the blood to the alveolæ.

The CO_2 will enter the pleural cavity from the tissue, but can never rise higher than 20 per cent, since this is the limit in the tissue, but the tension will tend constantly to rise above this, since the nitrogen is being absorbed.

204. Jaccoud. *Leçons de clinique médicale*, 1886-7, page 193.

The history of a case is given, a young man crushed between two wagons, compressing his left chest. Bloody expectoration, dyspnœa, pain in the side followed. In less than twenty-four hours he was brought to the hospital. No ribs were fractured, hence the lung was ruptured by the pressure. Temp. 38.8°. There was complete pneumothorax of the left side with tympanitic percussion note, amphoric respiration and voice sounds. There was no succussion splash, no metallic tinkling. At the posterior of the apex normal breath sounds could be heard. There was slight fever. Four days later fluid was present, which in four or five days was extensive in amount. This fluid seemed to ameliorate the symptoms. The patient felt much better.

There was some recrudescence of the fever. On the 17th day the signs were very different; the posterior thorax presented three superimposed zones, the superior and inferior were resonant, the middle dull. In the resonant zones was heard the respiratory murmur, in the middle ægophony, hence the effusion had become encysted. The remaining gas was at the apex. No succussion splash was obtainable. On the 27th day the dull zone was tapped and but 60 cc. of fluid obtained, and the writer decided the fluid was present in two sacs. The patient recovered.

The writer discusses this mode of recovery (by pleural adhesions) and the possible influence of these adhesions.

205. Harris. Cases of Spontaneous Pneumothorax. *St. Barth. Hosp. Reports*, XXIII, p. 33, 1887.

Case 1 was a man 32 yrs. old, with a history of constant exposure and chronic cough, who was treated for pleurisy with effusion of the left side. One day he had a severe fit of coughing, with feeling of collapse, and coughed up several pints of clear fluid. He was admitted to the hospital one week later with left-sided hydropneumothorax, hyperresonance, distant amphoric breathing, succussion, bell sound and an occasional metallic tinkle. He was also ill with acute articular rheumatism and cardiac disease. There were no indications of phthisis; the cough was ascribed to chronic bronchitis. The sequence suggested is, pleurisy with effusion, rupture of the lung by coughing, evacuation of the effusion through the lung.

Case 2.—This woman, 24 yrs. old, was evidently tuberculous, and developed suddenly pneumothorax when getting out of bed. The hyperresonance was extreme in extent, the apex beat was 1½ inches outside the left nipple, the liver reached the umbilicus. The breathing was distant amphoric, the coin sound and metallic tinkling were present. The onset was with extreme collapse and high fever. There was urgent dyspnœa.

Case 3 was a woman, 22 yrs. old, with phthisis, admitted with left-sided

pneumothorax which had had a sharp onset two weeks before. The distension was not extreme nor were the displacements. The breathing was loud and amphoric. The fistula was found large and open.

Case 4.—Course uncertain.

Case 5.—Tubercle bacilli in sputum gave diagnosis of cause.

"In conclusion I may remark that in my experience the bell sound is generally absent in cases of pneumothorax *at first*, and that it is then generally heard over wide areas. Later on the sound becomes more localized in extent and can be heard only on very careful and minute percussion with the coins over the whole of the chest. Not infrequently, however, the bell sound may be replaced, as the case progresses, by some less clear sound, which is almost exactly like that made by a hammer striking upon an anvil. I do not know whether this sound has previously received such a name, but I always call it the anvil sound."

206. **Hall (F. de Havilland).** On Cases of Pneumothorax in Persons Apparently Healthy. *Trans. of the Clin. Soc. of London*, 1887, p. 153.

Hall reports five cases, and adding these to West's twenty-one and Whipham's five proceeds to analyze these thirty-one cases.

Only two were women. Hall is confident that there is an antecedent strain, which, however, may precede by a few days. He discusses an old pleurisy, or latent tuberculosis, or emphysema, as the cause, and seems to think all three are possible.

He arranges in tabular form these additional ten cases.

207. **Gilbert.** Note sur les vergetures du thorax. *Arch. gén. de Méd.*, Vol. 159, p. 685, 1887.

The author, presenting a case of pleurisy presenting weals, reviews the literature of the subject, and concludes that in the young various pleuropulmonic diseases, among which is tuberculous pneumothorax, can cause weals, due to vulnerability of the skin and extreme distension of the chest.

He quotes Thoan's case of a young man, 17 yrs. old, with pneumothorax following caseous pneumonia, the left side of whose thorax from the spine to the sternum showed nine rows of weals parallel to the ribs, and about 1 cm. in diameter. (The description given answers well to liniæ atrophicæ).

208. **Augry.** Pneumothorax Caused by the Rupture of Emphysematous Vesicles in the Course of Chronic Pulmonary Emphysema. Paris, 1887.

Augry's conclusions are, that pneumothorax can follow the rupture of emphysematous vesicles; that such cases are rare; the symptoms are those of pure pneumothorax without fever or fluid exudate, and that they recover as a rule after or without puncture.

The writer collects seven cases from literature and reports two. All were men of advanced years with a history of hard work and all the other conditions favoring emphysema. The onset followed in some cases an effort, in others ordinary exertion, walking, and others while at rest.

In some cases it was sudden, with alarming symptoms; in other cases more gradual, with local symptoms.

Three cases were fatal, death being due to double pneumothorax or to the emphysema, the other lung not being able to do the work of both. The pleura was normal in these cases. The other cases recovered in from one to two months.

The characteristic clinical symptoms of such cases are the absence of fluid and of fever, since air alone is not an inflammatory agent and is sterilized by its passage over the moist mucous membrane of the respiratory tract. The air is absorbed in a few days or weeks. The danger is mechanical or due to the insufficiency of the remaining lung. The amphoric respiration and the metallic tinkling occurred.

208a. **Daland.** A Case of Pyopneumothorax of Nine Months' Duration. *The Transactions of the Philadelphia County Med. Soc.*, June 22, 1887.

The patient, a man 32 yrs. old, who had suffered from phthisis of the right lung for 22 months, at the end of which time sudden severe pain in the right chest, cough, high fever, and intense orthopnœa were considered to mark the ulceration into the pleural cavity. He was admitted to the hospital about six months later with all the signs of pneumothorax: tympany above, with movable dulness of four inches, metallic tinkling, amphoric breath sounds, the succussion splash, and bell tympany, displacement of liver and heart. Sixty-eight ounces of sero-pus were removed. Ten months later ten ounces were removed, and two months later the same amount of pure pus. The condition had improved a little four months later, still. (The condition continued for several months after the report of the case.)

209. **West.** Pneumothorax. *Brit. Med. Jour.*, August 20, 1887.

Excluding tuberculosis, gangrene, injury, and some other diseases leaves about 2 to 3 per cent of the cases due to various causes. Two forms of pneumothorax belonging to this group he will discuss. These are, first, the pneumothorax of healthy persons; second, that in which the pleural cavity has been laid open by injury. It is hard to prove a lung was normal, and the first question is, could a healthy lung be ruptured. The subcutaneous emphysema following paroxysms of coughing in whooping-cough, and the strain of parturition prove this possible, but pneumothorax rarely results, since the pleura is not ruptured, the air dissecting its way to the mediastinum. West quotes the work of Champneys, Wilks and Moxon, Money and his own, on the force required to burst the adult lung, finding a pressure of 2½ to 4 in. Hg., and for a perfectly normal lung probably more is necessary. He gives protocols of six experiments on this point. But a person could not exert a pressure of 8 to 9 in. Hg., hence a local lesion may be assumed if the pleura ruptures; hence the occurrence of pneumothorax in parturition or whooping-cough is exceedingly rare.

Again, in the cases of patients with pneumothorax who were considered healthy there is often no history of great exertion; in some cases it

developed during sleep, or while sitting still or walking quietly, and if exertion did cause it, it was usually not a great one. Hence the lungs were probably not truly sound. West gives interesting illustrations to show that lesions even tuberculous may exist, not be detected, and cause pneumothorax which gets well.

Pneumothorax due to an injury laying open the pleural cavity.—Sometimes the lung may be seen in the wound and yet the lung does not collapse; e. g., in one case the wound of the lung was three inches long, yet no pneumothorax; recovery followed. In other cases the lung is torn by a fractured rib, yet no pneumothorax results. West cites the case of a woman, several of whose ribs were broken, subcutaneous emphysema developed, but no pneumothorax. At autopsy the lung was found torn over an area of about two inches in diameter, but still the pleuræ were in contact and there were no adhesions. Such cases are not rare. The explanations given are, the obliteration of the pleural cavity by a former pleurisy, or the rapid absorption of the air, but these are not sufficient to explain clinical and pathological findings. To overcome the elasticity of the lung, which exerts a force of 7 mm. Hg. to collapse the lung, West considers there is a cohesion of 12.5 mm. between the pleuræ. This he determined by testing the cohesion of peritoneal surfaces, which experiments he considered conclusive. "Pneumothorax can therefore no longer be regarded as a condition to which there is an inherent tendency in the human body, but on the contrary as a condition brought about by the forcible separation of the pleural surfaces . . . and considering the frequency of subcutaneous emphysema and the rarity of pneumothorax in simple fracture of the ribs . . . the force required to distend the subcutaneous tissue must be less than that required to separate the layers of the pleura." This cohesive force he considers important in the physiology of joints, the pericardium, etc.

The intrapleural pressure in pneumothorax.—West quotes Donders' work concerning the elasticity of the lung, discusses the respiratory oscillation, and illustrates it by the pressures found in seven cases of pneumothorax. The displacements do not require positive pressure for their production. He doubts that increased tension can make the percussion tone muffled.

After discussing the course and prognosis of the disease he considers the methods by which the lung re-expands. If fluid be present it is removed by absorption, but the disappearance of gas is not so easy to explain. Simple diffusion will not do it, and Gréhaut showed that there was little interchange of gas through the pleura. The O is easily absorbed. The CO_2 is not as abundant as would be expected if it were exchanged for O volume for volume, but this interchange explains the initial absolute increase and absorption then reduces its amount. The N is later absorbed.

210. **Bouffaré.** Contribution à l'étude du pneumothorax de cause interne et de son prognostic. Paris, 1887.

The conclusions of this thesis are:

1. The prognosis of pneumothorax depends:

(a) On the cause producing it. It is more grave in advanced tuberculosis, following empyema and gangrene of the lung, than in emphysema or early tuberculosis.

(b) On the condition of the respiratory organs at the time of its production. If both lungs are involved asphyxia is inevitable.

(c) On the presence or absence of effusion. The pure pneumothorax is least dangerous, then hydro-, and lastly pyopneumothorax.

(d) A case of closed pneumothorax is less dangerous than one of open.

(e) On the extent of the condition, partial pneumothorax being least dangerous.

2. The tuberculosis does not seem arrested by this condition. If it does recede the general condition of the patient will explain the improvement.

An exception may perhaps be made in the case of hydro- or pyopneumothorax, since the resulting interstitial pneumonia may be able to check the tuberculous process.

These conclusions are based on the study of a few cases quoted from literature and a review of literature.

211. Bouveret. SUR LE PNEUMOTHORAX SUFFOCANT; PATHOGÉNIE. TRAITEMENT PAR L'ÉTABLISSEMENT D'UNE FISTULE THORACIQUE PERMANENTE. *Lyon médical*, Vol. LIX, p. 609, 1888.

The common form of pneumothorax begins suddenly with distressing symptoms. But soon a calm follows. The gas causes pleurisy, hence soon an exudate is added to the gas.

There is another form, happily rare, described as valvular pneumothorax, but for which a better name would be "suffocative pneumothorax," in which the symptoms, dyspnœa, etc., increase in severity till death.

CASE 1.—A tuberculous man, 24 yrs. old, in whom left-sided pneumothorax developed suddenly. The dyspnœa increased more and more till the following day asphyxia was feared, and was relieved at once by puncture with a cannula, which was left in the chest. He died that night. Advanced tuberculosis of both sides found. The perforation was narrow and sinuous.

CASE 2.—A man, 18 yrs. old, tuberculous, in whom right-sided pneumothorax rapidly developed, with great dyspnœa. The paroxysms of cough were severe and frequent. He died that night. The perforation was oblique, very narrow and sinuous.

Such are the suffocative forms. The compression is exerted on the sound lung, which in these cases showed as a result atelectatic areas.

How can this positive pressure in the pleura arise? He studied the literature on that subject and found the statement common that the air entered during inspiration; but that could never give a positive pressure. It is the expiratory efforts, and especially the cough, that forces in the

air which raises the pressure. And this is possible when the fistula is closed during inspiration and opened only by a cough. Such may be the case when the orifice has a membranous valve, or when the perforation is narrow and sinuous with collapsible walls. In such cases the cough is dangerous, and the pressure developed compresses not only the collapsed lung but the heart blood vessels and the lung of the other side.

There are then two forms of pneumothorax, the (1) open and the (2) suffocative.

To combat the cough in the latter, morphia is not enough. There must be a permanent fistula, either pleurotomy or a permanent cannula. Both are open to objections, since the external air may more easily infect the pleural cavity than that through the lungs. He prefers a cannula and proposes one. He advises its use even in cases of advanced tuberculosis.

Bouveret, according to Bard, was the first to reject Weil's explanation of valvular pneumothorax and to emphasize the importance of cough, which forces the air through a narrow sinuous fistula acting as a valve.

212. Piqué. The Chest. *Dict. encyclop. des. Sc. Méd.*, 2d Series, Vol. 26, p. 481, 1888.

Dolbeau showed that alone it is not enough that the pleura be opened. An incision one centimeter long may be made and the lung will not collapse. The two layers of the pleura glide easily on each other and yet resist a force tending to pull them apart, like two plates of wet glass. Smith showed the importance of the position of the wound.

Piqué considers the collapsed lung to take no part in respiration, if the chest be opened, and that air is forced out from the pleural cavity in expiration by the movement of the chest wall, this expulsion being still more favored by the fact that at the moment of expiration a part of the air in the good lung dilates the wounded lung.

213. Galliard. Du pneumothorax simple, sans liquide, et de sa curabilité. *Arch. gén. de méd.*, I, p. 275, 1888.

Introduction.—He reviews the work of Demarquay and Ewald on the absorption of gas in the pleura.

Physiological Pathology.—Animal experimentation shows that it is hard to produce pneumothorax by incision or puncture of the chest, since there exists a physical cohesion between the pleura. Pneumothorax may be due to rupture of a bleb.

The fistula may be obliterated by a false membrane at the orifice, or by cicatrization.

He then divides the cases of simple pneumothorax into:

1. Those occurring accidentally and concerning which we do not know seat or character of lesion, which recover.

2. The cases following some definite disease.

He proposes to study the former.

He collects from literature "the cases of simple accidental pneumothorax, following an effort, or not following an effort, which are spontaneous, in persons presumably healthy, and which recover in a few weeks." He gathers together twenty-two cases: 1, Rilliet and Barthey; 2, Dowell; 3 and 4, Thornburn; 5, Bodenheimer; 6, Vogel; 7, Abeille; 8, Oppolzer; 9, Renault; 10-11, Wilks; 12, Church; 13, Bojaczinski; 14, Bull; 15, Heitler; 16, Vinay; 17-20, Havilland Hall; 21, Galliard; 22, Ranking.

Good description of each case is given.

Page 441. Galliard now considers:

(a) Tuberculosis. Rupture of a cavity or of a softening tubercle, followed by simple pneumothorax. He quotes: 23, Biach; 24, Culmann; 25, Barrier; 26, Legendre.

(b) Emphysema of the lung causing simple pneumothorax. Of these he found: 27, Sanné; 28, Rheder; 29, Dittrich; 30, Gelmo; 31, Andrae; 32, Heitler.

Efforts of labor have often caused emphysema, but not pneumothorax.

(c) Other lesions of the lung. The symptoms of simple pneumothorax are now discussed, the pain at onset, and the constant dyspnœa. Cough is absent, unless a concomitant bronchitis be present. Fever never is found. The physical signs are now discussed, the hyperresonance and the absence of the respiratory murmur; but Galliard adds another, absence of vocal fremitus, which constitutes the diagnostic triad. The other auscultatory signs are found often, but may fail, and among these he mentions metallic tinkling, although others think this means fluid. The Hippocratic splash is always absent, the most important point in the diagnosis of this form.

He then discusses the diagnosis of the origin, the situation of the perforation, the question whether open or closed, etc.

Prognosis.—Galliard quotes the cases of: 33, Ferrari; 34, Biermer; 35, Förster, where there was some fluid but recovery.

He advises morphia, oxygen inhalations, puncture if the pressure gets high, but he doubts its value.

Conclusions.—There is a variety of unilateral pneumothorax which usually follows an effort, and occurs in men from 17 to 37 yrs. of age in whom the existence of a pulmonary lesion cannot be proven. This form is total and is cured in four weeks or less. It is very rarely caused by tuberculosis, the only cause which can be assigned for most being the rupture of a group of emphysematous vesicles.

214. Bruen. Percussion as a Means of Diagnosis of Pneumothorax. *The Medical News*, Dec. 1, 1888.

One case is reported. The writer emphasizes the percussion sign of Trousseau (bruit d'airain) as of special value. He also recommends the introduction of sterile air as the pleural fluid is withdrawn, which by keeping the lung at rest favors cicatrization and the cure of tuberculous lesions.

215. Marion. Pneumothorax with Autopsy, in a Subject with Extreme Retro-lateral Curvature of the Spine. *Boston Med. and Surg. Jour.*, May 31, 1888.

The patient was a man, 43 yrs. old, kyphotic from childhood, who developed pneumothorax following pleurisy. Operation was performed and a drainage tube kept in the wound for three months. Then followed five months of comparative good health, the wound being closed. He died later of pyæmia. He considers his a case of pneumothorax following empyema or simple acute pleurisy.

216. Widal. Pneumothorax. *Dictionnaire Encyclopédique des Sciences méd.*, 2d Series, Vol. 26, 1888.

This article will give a good idea of the subject to this date. Widal defines the disease as the presence of air or any other gas in the pleura. As a rule fluid effusion is also present; sometimes the gas precedes and acting as the inflammatory agent causes the effusion, or the latter may precede, or both may develop simultaneously.

Etiology.—The gas may arise in the pleura or from the outside.

1. In the pleura.

Essential. Graves believed in this, due to a subacute pleurisy, but of which no anatomical lesion could be found.

Due to the decomposition of pus. Jaccoud admits, Béhier denies its possibility. Thus this form is not well proven. Béhier claims air necessary for decomposition.

Gases contained in the fluid may be set free and cause pneumothorax. This is the case after aspiration which diminishes the pressure on the liquid in chests where the adherent lungs cannot expand and the chest wall contract to keep the tension the same. (Lee.)

After empyema perforating internally or externally, and after operations.

After the rupture of interlobular pleurisies.

2. Diseases of the lungs.

Tuberculosis. Acute or the acute recrudescence of chronic cases in which cases the pleuræ are not adherent.

Emphysema. In this connection Widal mentions two cases, a military officer, emphysematous, in whom it developed while he was going down stairs, and a trumpeter, in whom pneumothorax developed while he was blowing the trumpet. Also Desnos' case is cited, in which a purulent exudate followed the pneumothorax due to effort.

Pulmonary gangrene (cases of Cruvielhier, Monneret, Marais, Biach).

Abscess of the lungs (cases of Gunsbourg, Dalmas).

Pulmonary infarction (Carswell).

Dilatation of the bronchi (Moher, Taylor).

Capillary bronchitis of infants (Vallin).

3. Following lesions of neighboring organs.

(a) Chest. Rupture of the œsophagus in cancer or awkward catheterization (Boerhaave, Montard, Martin).

Peripleural abscess.
Abscess of the bronchial glands.

(b) Abdomen. Perforating ulcer or cancer of the stomach.
Abscess or hydatid cyst of the liver.
Abscess or hydatid cyst of the kidney.
Caries of the ribs or sternum.
Perforation of the duodenum or transverse colon, by an abscess or a round worm (Bouchard, Eisenlohr, Cossy, Leyden) or appendix abscess.

These cause pseudo-pneumothorax in case the diaphragm is not perforated.

4. Traumatic pneumothorax.
Violent coughing (Ramskiel).
Forced marching.
Fall.
Use of wind instruments.

Probably these alone cannot rupture the lung; either tuberculosis or emphysema must be present.

Pathological Anatomy.—The writer discusses the seat of perforation, and the valvular, open or closed character of the fistula, gas analysis, etc.

To explain difference in fluid exudates Vieuille supposed that O is irritating, N and CO_2 not, hence when the fistula is short and access of air easy one finds empyema; when long, hence less O, the exudate is serous. The sterilizing effect of the bronchi was shown by Lister, Strauss, Dubrenith.

Symptoms.

Physical Signs.—The dilatation of the chest need not be true distension, but apparent since there is inability to expire (Castelnau, Béhier).

Percussion when tension is high gives a higher pitched tone. At the level of the spine where the lung lies the note is duller. The bruit d'airain depends on the tension of the gas, being absent if this is high and reappearing as it diminishes. The respiratory murmur may fail or be amphoric with a metallic quality.

Diagnosis.—A very good discussion. One must exclude large cavities where even the Hippocratic succussion may be present. But the vocal fremitus will be present or increased, the chest will show no dilatation, the neighboring organs will not be displaced, cavernous râles will be present and Wintrich's change of pitch will be demonstrable.

In open pneumothorax the chest will be slightly dilated, immobile, metallic respiration at a maximum, the displacement of organs at a minimum, the metallic tinkling and amphoric breath sounds are present and CO_2 will be under 5 per cent.

In closed pneumothorax the chest is immobile, the distension great, the dyspnœa excessive, the metallic sounds less than in the open, the amphoric resonance diminished or absent, and the neighboring organs considerably displaced.

The valvular lies between these two.

Prognosis.—Czernicki, Hérard. Pneumothorax aids the cure of tuberculosis.

Treatment.—Weil, Ewald.

Widal thinks the fistula will close in from eight to fifteen days.

217. Moizard. Pneumothorax et antiseptie pleurale. *Gaz. des Hôp.*, Paris, Vol. 61, p. 865, 1888.

Following Potain's method, the writer treated two cases by injecting into the pleural cavity iodine solutions (30 and 70 gms.). The results were satisfactory.

218. Potain. Des injections intra-pleurales d'air stérilisé dans le traitement des épanchements consécutifs au pneumothorax. *Gaz. des Hôp.*, Vol. 61, p. 451, April 26, 1888.

Potain advises to remove if necessary all the liquid exudate of a pneumothorax and substitute sterile air for the same. The air is passed through cotton to remove the germs and is non-irritable to the pleura. By so doing one avoids the dangers resulting from the presence of a large effusion, or the rapid evacuation of a large effusion. One avoids the need of frequent punctures and obtains slow progressive distension of the lung. The rest which the diseased lung gets seems to favor cicatrization and healing of the tuberculous lesions.

219. Gabb. Recurrent Pneumothorax. *Brit. Med. Journal*, July 28, 1888.

A woman 56 yrs. old, who had four attacks of pneumothorax at intervals of six, two and five years. The right was the affected side each time; there was evidence of some pleurisy, but little fluid. The recoveries were rapid. Bramwell, who saw the patient in two of the attacks, considered it idiopathic. Slight exertion was on one occasion assigned as the cause. No disease of the lung was recognized.

220. Riedinger. Verletzungen und Chirurgische Krankheiten des Thorax. *Deutsche Chirurgie*, Vol. 41-44, p. 63, 1888.

As a complication of fractured ribs the writer evidently considers a severe pulmonary wound necessary to produce pneumothorax. The condition may appear several days after the fracture. Billroth saw pneumothorax in 4 of 58 non-fatal cases of fractured ribs, and considered it rarer than was generally supposed.

He mentions Hird's case, in which the sequence was; fractured rib, hæmoptysis, pneumothorax, and later pyothorax; and Aschenborn's, with pneumothorax and pleuropneumonia.

221. Turner. Pneumothorax and Hydro-pneumothorax following Fracture of the Ribs; Remarks. *Lancet*, 1889, p. 479.

The writer's report is occasioned by West's inquiries of surgeons con-

cerning the occurrence of pneumothorax as a result of fractured ribs. The writer states that emphysema is a common result, yet it is unusual to find clinical accounts of cases of pneumothorax. The *St. Thomas Hospital Reports* from the year 1877 to 1887 contain 237 cases of fractured ribs, with 28 deaths. Of these there was emphysema in 25, hæmoptysis in 28, but in only 4, and these all fatal, was pneumothorax found, "and it was accompanied in every instance by hæmothorax, in three by emphysema, and later by pleurisy in one. All of these were the subjects of severe injuries."

He reports one case with recovery.

His conclusion is that "cases of pneumothorax . . . are comparatively rare, seeing how common an injury is fracture of the ribs, and how common the complication of emphysema and hæmoptysis, showing that the lung has been injured."

222. **Sehrwald.** Zum Atmungmechanismus bei offenem Pneumothorax. *Deuts. med. Wochensch.*, Aug. 22, 1889.

His conclusions are:

1. The effect on the respiration of a unilateral pneumothorax opening externally depends not on the absolute size of the opening, but on the relation of its size to that of the bronchus of that lung.

2. The second determining factor for the ventilation of the sound lung is the nature of the mediastinum. If it is very thin and distensible each inspiration will aspirate it so much to the good side that it hinders the dilatation of that lung. If it is thick and non-pliable it serves as a firm vantage point for the distension of the sound lung and makes possible a sufficient gas exchange.

3. For this reason the young and persons with a normal mediastinum endure pneumothorax worse than those in whom it has become a thick, stiff membrane. The formation of thickenings in the pleura is in these cases an advantage to the individual.

4. The functional condition of the mediastinum is also of great importance. In inspiration the descent of the diaphragm and the enlargement of the sagittal plane of the thorax puts the mediastinum on the stretch, thus aiding the sound side.

5. In open pneumothorax this factor is still more worthy of notice, since here the inspiratory movements of the chest wall and the diaphragm increase four or five times.

6. To force the collapsed lung to again take some part in respiration it is well to close the thoracic opening at the end of forced expiration with a tight bandage.

To close a double pneumothorax at the height of inspiration renders ventilation of the lungs impossible, but if at the height of expiration, the pressure on expiration is 0, during inspiration negative, hence usually there is sufficient ventilation.

These conclusions are based on experiments, the observation of one case and the study of literature. Unfortunately the writer gives little idea of the number or technic or character of the first.

223. Ewart. Open Pneumothorax. *Lancet*, Nov. 16, 1889, II, p. 1098.

This case of tuberculous pneumothorax is mentioned. At first the manometer indicated open pneumothorax.

The gas analysis gave O = 3.5 per cent; CO_2 = 17.4 per cent; N = 79.1 per cent, which indicates a closed fistula.

224. Nichols. On Pneumothorax in Children. *Homeopathic Journ. of Obstetrics*, XI, p. 201, 1889.

The rarity of the condition in children is mentioned. The writer says in speaking of pneumothorax in general: "Hillis and Lower, two English physicians who flourished towards the close of the seventeenth century, were probably the first who described a rupture of the pleura followed by the passage of air into the pleural sac." "The course of pneumothorax in children is always more rapid than in adults. . . . But Rilliet and Barthez report a striking case in which recovery ensued after the positive signs of pneumothorax had persisted for twenty days in a boy of three years of age."

No case is reported.

225. West. Recurrent Pneumothorax. *Brit. Med. Jour.*, Jan. 12, 1889.

A man 22 yrs. old, on getting out of bed, had a sudden attack of dyspnœa; no other symptoms; no previous ill health. Simple pneumothorax of the right side was found and rapid recovery followed.

About seven months later the trouble recurred. He felt while quietly walking a pain above the right clavicle, he felt a bubbling sensation, and cough, but was well in about two weeks.

Three months later a similar attack came on during a football game. Pneumothorax was diagnosed ten days later. He grumbled at staying in bed.

The interesting points are its latency (of the first attack), the fact that he was a healthy man, and that he had surely two and probably three attacks.

226. Thompson. Further Observations upon the Pleural Cavity. *Researches of the Loomis Laboratory of the Univ. of the City of New York*, No. 1, 1890.

This note is very brief. He mentions previous experiments, and a glass window which he put in the chest wall of a dog for over two months, allowing the intrapleural conditions to be well followed.

227. Dwyer. The Mechanism by which the Lung is Reinflated. *Researches of the Loomis Laboratory of the Med. Dep. of the Univ. of the City of New York*, No. 1, 1890.

The few but well-selected experiments demonstrate that in pneumothorax the collapsed lung is expanded by the air forced into it from the sound lung. By a few forcible expiratory efforts a dog can rapidly expand a collapsed lung. This cannot take place if by free incision into the trachea the effect of the constriction of the glottis be prevented.

228. Northrup. The Effect of Opening the Pleural Cavity. *Researches of the Loomis Laboratory of the Univ. of the City of New York*, No. 1, 1890.

Experiments were performed to test West's idea of the cohesion of the pleuræ. The writer found no evidence for this. The pleura was carefully opened by a pin-hole, care being taken not to separate the two serous leaves. The lungs collapsed at once.

In another experiment the effect of double pneumothorax was determined. If the openings be closed the dog was little disturbed. With open pneumothorax on one side, closed on the other, there was little discomfort, but both open caused urgent dyspnœa. Through a glass window in the chest wall the lungs could be watched to expand.

229. Leyden. Über Pneumothorax Tuberculosus nebst Bemerkungen über Heilstätten für Tuberculose. *Deut. med. Wochenschr.*, Feb. 13, 1890.

The remarks are of a rather general character. The speaker (at the Verein f. inn. Med.) showed one recovered case, and reported others, three of seropneumothorax, and three of pyopneumothorax; one, a cured case, he demonstrated.

230. Liebermeister. Pneumothorax. *Deut. med. Wochenschr.*, May 1, 1890.

The paper is a general review of the subject without the report of cases, work, or references to literature. He states that Leichtenstern proved on one case both ante and post mortem that the metallic tinkling may be caused by fluid falling from fibrinous strands in the upper part of the chest into the fluid below, after changing the position of the patient.

231. Williams. Pneumothorax Occurring in the Course of Phthisis. *New York Med. Jour.*, March 22, 1890.

The patient, a man 29 yrs. old, phthisical, was found suffering from pneumothorax on the right side. Owing to his dyspnœa, a trocar was inserted and the Allen surgical pump attached, and since the chest filled in ten minutes after the pumping stopped, and he would beg its renewal, the pumping was kept up four days and ten hours. He lived ten days longer and died from the phthisis. The right lung was tuberculous, but no sign of pneumothorax was present.

232. Knight. Case of Recurrent Pneumothorax in a Boy. *St. Barth. Hosp. Reports*, 1890, p. 294.

Case of a boy, 16 yrs. old, with tuberculous symptoms for three years. Admitted with pneumothorax on the right side. He states that one year and eight months ago he had a similar attack. Eight months later, when seen again, there were no symptoms of pneumothorax.

233. Waller. Pneumothorax in an Apparently Healthy Young Man without Realized Injury; Tapping; Recovery. *Lancet*, Feb. 8, 1890.

Young man, aged 16 yrs., had, following a cold of a little over a week's duration, right-sided pneumothorax (not with a sudden onset), accompanied with fever (102°). Aspiration allowed air but no fluid to escape. The patient recovered from the pneumothorax (in about five weeks), but "subsequently he had a slight relapse in the way of cough and feverishness, necessitating two days' rest in bed."

234. Lasius. (A Case of Bilateral Pneumothorax.) Leipsic, 1891.

In this thesis is reported the case of a woman, 37 yrs. old, who suffered from tuberculosis with cavity formation of the right apex, which process dated back one and a half years. Suddenly she had intense dyspnœa and the sensation of suffocation; right-sided pneumothorax was found. The following day she was confined, and three days later the signs on the left side indicated pneumothorax. Six days later she died. At autopsy the diaphragm on the right reached the fifth rib, but on the left was convex downwards. In the thorax was found bilateral pneumothorax. (For a description of the conditions on the right side and a discussion of their relation to the physical signs the reader is referred to the thesis.)

235. Lundie. A Case of Spontaneous Pneumo-thorax and Pneumopericardium. *Edinburgh Med. Jour.*, Sept., 1891.

The case was a young man 20 yrs. old, who had had slight symptoms of tuberculosis for two or three years, but the symptoms were very slight. One day while sitting quietly he felt a sudden severe pain in the region of the left shoulder blade, followed by breathlessness and distress on the slightest exertion. Left-sided pneumothorax was found, with the heart displaced backward rather than to the right. He recovered without signs of either inflammation or effusion. In about six weeks he returned because of pain and sense of splashing in the left side, from which he recovered. The cardiac signs were especially interesting, the motility of the heart and its "water-wheel sound."

236. Aron. (Concerning an Attempt to Measure the Intrapleural Pressure of Living Persons.) *Virchow's Archiv*, 126, p. 517, 1891.

This seems the first successful determination of intrapleural tension in normal man. The review of literature on that subject is good. (The observations are again reported in the author's publication of 1902, to the abstract of which the reader is referred.)

237. Gilbert and Roger. Étude expérimentale sur le pneumothorax et sur les reflexes d'origine pleurale. *Revue de Médecine*, 1891, p. 977.

Weil omitted the study of the respiratory changes during the production of a pneumothorax, the state of the heart and the blood pressure either at that time or later, nor did he study the respiratory phenomena in double pneumothorax.

I. *Experimental Pneumothorax.*—The authors worked on dogs, measuring respiratory movements and blood pressure, at the production of pneumothorax. At the moment when this condition is produced the respiratory rhythm is much disturbed, there being marked inspiratory movements, temporary, then the curve falls to its former regularity. The blood pressure is little changed, or in some cases there is a drop in pressure followed by slower, larger beats, but soon resuming the former rhythm. After the condition is established in the case of open pneumothorax the rate is faster, inspiration deeper and on the line during expiration can be seen a secondary wave caused by the air leaving the thoracic wound.

In closed pneumothorax the rate is slower than normal, and the amplitude of the movements greater, but less than in open pneumothorax. The line of inspiration is almost vertical, expiration sudden.

Double pneumothorax closed causes an increased rate, the waves being of less amplitude and dicrotic. If now one side be opened the amplitude of movement increases at once; if both sides are open the amplitude increases still, now reaching three times the normal range.

II. *Reflex disturbances of pleural origin.*—These were studied first by Roger. They are seen in old pleurisies, after the injection of fluid into the pleura, or after operations on the pleura. They are transitory as a rule. They produce a fainting spell, a convulsive attack, or transitory paralysis; more rarely are they permanent (he reviews the figures on their occurrence), a paralysis or hemichorea. They found in two animals similar results of intrapleural injections, but could not produce them at will. One was a guinea pig, convulsions following the injection of a culture of cholera spirilla, the other a dog, into whose pleura a little metallic mercury was injected and which showed rhythmical contractions of the face and jaw. These two animals are described at length and the literature of the latter (tic) given, which they consider a reflex through the X and V cranial nerves, although it remained after section of the vagus nerve.

Good tracings (brief) of respiration and pulse are given.

238. Kreps. Über die Athmungsbewegungen bei den verschiedenen Formen des Pneumothorax. Dorpat, 1891.

This student in the introduction of his thesis calls attention to the priority which Unverricht, under whose direction he worked, claimed for the recognition of the importance of valvular pneumothorax, and he begins by quoting from Unverricht the description of the three sharply defined groups, open, closed, and valvular.

Kreps worked on narcotized and unnarcotized dogs, and discusses the influence of morphine and tracheotomy on the respiration. By a cannula

provided with a stop cock thrust into the chest he produced the various sorts of pneumothorax. For the valvular form he used a mercury valve. A special apparatus (by Unverricht) registered the motion of the chest wall, depth of respiration being thus measured.

Open pneumothorax produced excursions of the thorax of increased rapidity and depth, the extent of increase depending on the width of the fistula. The frequency did not change in direct proportion to the width, but the depth always did, and he discusses the reason.

In the transition from open pneumothorax to that closed during expiration both factors increase, frequency and depth of respiration. In the transition from a closed to an open one the frequency increases, but the depth increases, unless a great deal of air had been introduced, in which case the depth increases.

Closed pneumothorax produced by injecting air into the pleura, as a rule causes diminished frequency and increased depth of respiration. After a certain limit has been reached the depth diminishes.

In pneumothorax closed during expiration the rule is an increase in rate and depth; the transition from a valvular to a closed caused diminished frequency and depth.

Valvular pneumothorax causes an increase in frequency and depth of respiration until a certain point is reached, beyond which the depth is diminished in proportion to the amount introduced.

Protocols of the work on seventeen dogs are given.

239. Szupak. Experimentelle Untersuchungen über den Pneumothorax. Dorpat, 1891.

This work was done under the direction of Unverricht to confirm Wintrich's work concerning the inflammatory action of air on, and its absorption by, the pleura.

Experimental Section.—Twenty-three dogs were experimented on. The pleura was opened aseptically, a definite amount of air injected without injuring the lung, and the wound then closed. The animal was later (2 days) killed and the pleuræ inspected. The amount of air introduced was proportional to the absorbing surface. Wintrich had studied the normal pleura, so Szupak does the abnormal, a pleurisy being produced by the introduction of a solution of lunar caustic and the gas introduced twenty-four hours later. He thus studied serous pleurisy, but was unable to produce the purulent variety, the experiment failing or the animal dying.

The animal was killed at the end of the experiment by bleeding it, and as a side problem the writer determined that dogs could thus be bled on an average of 53.75 gms. per 1 k. of body weight. After death the trachea was tightly clamped to prevent collapse of the lung, and the chest opened under water. Since the dog's mediastina often communicate, these cases must be excluded. He determined that in closed pneumothorax a considerable amount of air is in twenty-four hours absorbed on the normal side. He then tries the separate constituents of air (on

normal pleuræ?) and found that pure CO_2 and pure O are absorbed in the same time as atmospheric air, while in that same time more than half of pure N is unabsorbed, hence differing from Wintrich, who found pure N took the same time as air to be absorbed; but the author's result confirms most other workers.

In pneumothorax with serous pleuritis the absorption of air occurs always, but the time taken is proportional to the intensity of the inflammation, and the number and strength of adhesions that have formed.

A further result of this work is that the air in pneumothorax is not an inflammatory agent, nor does its presence initiate decomposition of an inflammatory exudate. A discussion of the effect of silver nitrate on the tissues follows.

Clinical Section.—The author will now see how much in harmony clinical observations are with his results.

He reviews cases of traumatic pneumothorax and other cases of pneumothorax with recovery to show the possibility of recovery and without any signs that pleurisy had resulted, but the time required for some varied from twenty-six days to two months (such cases without friction rub, or fluid exudate) while one case (Nonne) required but sixteen days, Bodenheimer's only six, but Adams' case three years. The difference in such cases he ascribes to the lung fistula. "In all cases in which the air absorption consumed more time than our experiments showed necessary, the diagnosis of a mechanically closed valvular pneumothorax is justified" (hence the air is renewed).

(That the influence of pleural disease on the time of recovery would agree with his experimental results scarcely needs attention.)

The review of such cases is particularly good.

240. **Zahn.** Über die Entstehungsweise von Pneumothorax durch Continuitätstrennung der Lungenpleura ohne eitrige Entzündung. *Virchow's Archiv*, Bd. 123, p. 197, 1891.

Such cases are very rare in literature, and but six with autopsy. The author will report six.

A. *Pneumothorax from the rupture of an emphysematous vesicle.*—In these two cases the lungs were emphysematous, and in one case at least coughing ruptured a bleb; in the other case there was probably combined vesicular and interstitial emphysema. This case he thinks unique since there was œdema of the chest-wall, due to the increased intrapleural pressure which obstructed the lymph flow. In literature he could find but four cases with autopsy in which there was anatomical proof that the pneumothorax was due to the rupture of an emphysematous vesicle and the overlying pleura.

B. *Pneumothorax due to the rupture of an interstitial pleural emphysematous bleb.*—Two cases are reported. No cases were found in literature. In this case the passage of air was indirect, from the lung to the interstitial tissue and thence to the pleura. The sign of this form is not alone the seat of the rupture, but the presence around

it of several interstitial emphysematous blebs. He is convinced that the majority of cases due to effort arise in this way.

C. *Pneumothorax due to the tearing of the pleura, besides old adhesions.*—One case is reported and Robertson's the only one found in literature (*Glasgow Med. Jour.*, 1888).

D. *Pneumothorax from atrophy of the visceral pleura.*—The condition was discovered at autopsy. The lungs were emphysematous. Seven perforations were found, but these were not tears, but round holes and looked as if made with a punch. Neither ecchymoses, interstitial emphysema, nor adhesions were present. The pleura was remarkably thin. He considers the perforations the result of pressure atrophy and disturbed nutrition.

In conclusion he states that therefore pneumothorax from exertion can arise in several ways; in the first methods, the production, is sudden, in the last, slow. "*a*" is the rarest, "*b*" the common method.

Of his six cases four were men, and this he thinks is due to the chronic bronchial catarrh which favors emphysema, and which is more common in men, and was present in five of these cases. Of the six cases, in three there was no pleurisy, in two a slight. The other case had pleurisy, not due to the air. In such cases the air is sterile, and in one case above with pleurisy the air entered so quickly that the filtration in the lungs was imperfect.

240a. Zahn. Nachtrag. *Virchow's Archiv*, Vol. 124, p. 265, 1891.

Another case of pneumothorax due to the rupture of a pleural interstitial emphysematous bleb is reported.

241. Glaeser. Pneumothorax mit besonderem Verlauf. *Zeitsch. f. klin. Med.*, Vol. XXI, p. 394, 1892.

The patient, a man 32 years old, was suffering from acute articular rheumatism, and suddenly developed left-sided pneumothorax. Recovery in about ten days. No exudate.

242. Senator. (A Contribution to the Knowledge and the Treatment of Pneumothorax With or Without Fluid Exudate: With Remarks Concerning the Operative Evacuation of Empyema.) *Zeitsch. f. klin. Med.*, Vol. 2, 1892.

Senator first combats the current opinion that the exudate in pneumothorax is, as a rule, purulent. He reports I. a case of chronic bilateral incipient bronchopneumonia; pneumothorax of the left side. Left-sided pleurisy. Aspiration of a serous fluid twice. Recovery.

II.—Right-sided pneumothorax in an apparently healthy person. Right-sided pleurisy, at first serous; after aspiration, it re-collected becoming purulent. Two evacuations by double incision and drainage. Death at the end of a three months' illness.

III.—Chronic left-sided pneumonia with bronchiectasis. Left-sided pneumothorax, pleurisy with sero-fibrinous exudate, fresh pericarditis, death.

He calls attention to the heretofore unnoticed frequent occurrence of bilateral pleurisy with unilateral pneumothorax, and also to the infrequence of putrid exudates in cases where the air has free entry to the cavity internally or even externally. The pleural exudates in children are more often purulent than in adults, which he ascribes to the greater vulnerability of the child's pleura. He reports a case illustrating the well-known fact that a pneumothorax may exist a long time without an exudate, also that in long standing pneumothorax the exudate present may disappear or at least diminish.

Case IV.—Pneumothorax of the left side in the course of a case of chronic caseous bronchopneumonia. Left-sided pleurisy with slight exudate, which, in spite of the long-continued presence of air in the pleura, was absorbed, and then collected again, etc.

But, although air itself is not an inflammatory agent, in the case of pneumothorax due to an external wound a pleurisy always results. For the explanation he thinks the temperature of the air of little importance. The filtering function of the lung he thinks important, removing the bacteria. But even in cases where a large cavity empties into the pleura, causing purulent exudate, it seldom becomes putrid, and this he ascribes to the high CO_2 content of the air in the pleura, which has an antiseptic action.

In favor of the importance of these two factors, the filtering power of the lung and the antiseptic action of CO_2, is the fact that when an empyema ruptures through a bronchus causing pneumothorax, putrefaction of the exudate usually results, since a large bronchus is opened hence less CO_2 and less filtering, but if it ruptures through the lung tissue the pyopneumothorax does not so often result.

The next question discussed is spontaneous pneumothorax, *i. e.*, without lesion of the pleura. Two possibilities only need be considered; first, the liberation from an exudate of gas after the pressure has been much diminished by aspiration (Bucquoy). This is hardly important. Second, pneumothorax from the decomposition of fluid. This is possible even in the absence of air as gas formation in other parts of the body (he cites his experience) shows, and is favored by the thickened condition of the pleura preventing absorption of the gas.

Concerning operative treatment of pneumothorax his conclusions are:

1. So long as it is pure pneumothorax, or with a slight amount of fluid, no operation is indicated. The removal of gas by puncture is harmless, but useless. The dyspnœa should be met by morphia, etc.

2. If exudate be present and there are no immediate indications, wait till the gas is absorbed, and only after this, or as severe symptoms appear, remove the fluid. If puncture shows purulent fluid incise at once; if serous aspirate, but the latter is indicated in purulent exudates of tuberculous cases, or when the lungs are not especially diseased. He next describes the technic of aspiration, etc.

243. Blumenthal. Experimentelle Untersuchungen über den Lungengaswechsel bei den verschiedenen Formen des Pneumothorax. Dorpat, 1892.

His work was done under Unverricht's direction. To determine the amount of air respired a Gasuhr of the Clegg variety connected with the tracheal cannula was used, well standardized. The dogs were narcotized with morphine.

By depth of respiration was meant the amount of air respired per respiration; by volume, the amount per minute.

The valvular pneumothorax was produced by the Kreps method, using a membranous valve. Open (glass cannulas of various sizes being used), closed, and those closed during expiration or inspiration, were the five forms studied. His conclusions, drawn by experiments on fifteen dogs, are the following:

1. In all forms of pneumothorax the rate of respiration is increased.

2. This is especially marked during pneumothorax closed during expiration, less if during inspiration. Between these extremes lies open pneumothorax. The pneumothorax, produced by injecting air, shows a slight increase in frequency directly proportional to the amount of air injected.

3. The volume of respiration increases in both forms of closed pneumothorax, and in open is diminished with increase in diameter of the fistula.

4. The volume of respiration increases only after considerable air has been injected into the pleural cavity, and then the increase is not great.

5. In open pneumothorax the volume of respiration varies inversely as the size of the fistula, and sinks below normal only when the latter is very wide.

6. In valvular pneumothorax the volume of respiration, and all other values, are greatly dependent on the resistance which the valve offers.

7. The depth of respiration is in all forms, in open, that closed during inspiration, that during expiration, about equally diminished.

8. In the pneumothorax caused by injecting air, this diminution is slight, until considerable is injected, then it agrees with the other forms.

9. The depth is in open pneumothorax approximately proportional to the size of the fistula; in 10 valvular cases it is most diminished, after a few breaths reaching only one-fourth the normal amount, and rapidly becoming 0.

The work seems carefully done, the literature is good, the technic well described.

244. Murdoch. An Unusual Case of Pneumothorax. *Medical Record*, July 9, 1892.

A girl, 15 years old, had a pneumothorax follow a severe fit of coughing. There were no symptoms of tuberculosis, no dyspnœa; recovery in four weeks. The unknown origin and lack of dyspnœa are the symptoms to which Murdoch calls attention.

245. Sunderland. Aspiration in Pneumothorax. *Lancet*, June 25, 1892 (Vol. I, p. 1419).

This is the report of a case developing in a case of phthisis. The patient was a young man, 32 years old, whose tuberculous history dates back at least seven months. The pneumothorax resulted from a severe fit of coughing, and the characteristic symptoms were present. He was relieved by aspiration (the distension of the left side was considerable) several times, the special indication evidently being cardiac weakness. Eleven months later he reported himself "well and strong."

The writer quotes several leading English authorities who advised against aspiration of air in such cases.

246. Munro. Pneumothorax. *Lancet*, August 13, 1892.

A case of this disease in a seaman, 24 years old, is described. Aspiration gave no relief. The disease was on the left side, tuberculous in origin, sudden outset. Death. Autopsy.

247. Galliard. Le Pneumothorax. *Bibliothèque Médicale* (Charcot-Debove), 1892.

This little volume, one of a series of such books, contains a very good review of the whole subject in its every aspect except the traumatic cases. It contains no new communication.

248. Nothnagel. Über Pneumothorax. *Internat'l klinische Rundschau*, 1892, VI, page 1337.

The patient, a man 38 yrs. of age, enjoying good health, was suddenly seized by a severe pain between the shoulder blades, followed by severe continuous dyspnœa and cyanosis. Such an attack in a patient with disease of the respiratory organs always suggests pneumothorax, but not always is this the cause, as in one such case in which the autopsy showed the rupture of the contents of a cavity into the bronchi. Hence physical examination is necessary. Each sign is then discussed. The cyanosis, unilateral distension (which means exudate, air or a tumor in the pleura), emphysema never obliterates the interspaces. The displacement of the heart; Hippocratic succussion splash which always means air and fluid in the pleura, that produced in large cavities never having the same intensity. He cites one case in which the stomach splash caused mistaken diagnosis, and the hyperresonant area discovered was found to be a large cavity which had just ruptured into the bronchi. The situation of such a cavity will aid diagnosis. If at the apex it is a point in favor of cavity rather than pneumothorax.

Page 1381. The percussion tone is loud with a metallic quality. This tone depends on the tension of the gas. When such a tone alters, becoming more tympanitic, it indicates a diminution in the amount of air, hence a good sign. The note is not tympanitic when the tension is increased.

The tactile fremitus is not absent, as is usually stated, only dimin-

ished, the vibrations being propagated through the air in the chest and in the chest wall.

The cause is then discussed. He cites one case of extreme emphysema with all the symptoms of pneumothorax.

The treatment is discussed, aspiration is advised if the asphyxia be extreme.

The prognosis is not so bad as formerly supposed.

249. Jamieson. Cases of Pneumothorax, illustrating the Causation and Mode of Occurrence. *Australian Medical Journal*, Aug. 15, 1893.

The writer says in Australia the rupture of an hydatid of the lung into the pleura is a well-recognized cause of pneumothorax, although not very common.

The three cases reported present little that is new.

250. Vickery. Cases of Pneumothorax. *Boston Medical and Surgical Journal*, July 20, 1893.

A report of ten cases, of various origin, with remarks especially on the latency of the disease, e. g., case VIII "had noticed a swashing sound in his chest for six weeks before he took to his bed; that is, he had been working as a hodcarrier for six weeks while suffering from hydropneumothorax."

251. Klemperer. Fall von geheilten Pneumothorax. *Deut. med. Wochenschr.*, June 22, 1893.

The case was presented before the Verein für innere Medizin. The man was a huckster, 21 yrs. old, who must yell much in peddling his wares. Suddenly he felt a sharp pain in the left side and fell unconscious. The cause was probably emphysema; there was no suspicion of tuberculosis. Perfect recovery in 3½ weeks. A discussion by Guttmann, Litten, Leyden and Lazarus follows.

252. Cnopf. Über Pneumothorax im Kindesalter. *Münch. med. Wochensch.*, Vol. XL, p. 123, 1893.

Biach's review of 918 cases of pneumothorax is first mentioned. Also West's figures and others. In childhood it is a rare disease. Lentz collected 35 cases in literature. The cause was:

Tuberculosis in 14; gangrene, 11; emphysema, 3; lung apoplexy, 3; fractured rib, empyema, hemorrhagic infarct, bronchiectasis, 1 each.

Steffen collected 16 cases with similar result. The cases are centripetal or centrifugal (i. e., according to the direction of the perforation). Cases due to lesions of neighboring organs are rare in children. The writer then reports a case.

Case 1.—The child was 2½ yrs. old, suffering from diphtheria of three days' duration. The asphyxia rendered tracheotomy necessary. The condition improved much, and the child slept well that night, but the next

morning critical symptoms of laryngeal stenosis necessitated the use of the bronchial spoon, but without result. The child died that day.

At autopsy a remarkable picture presented itself. Both lungs were well aerated, but collapsed so that the heart and large vessels were uncovered, and both bases were distant about four fingers' breadth from the diaphragm. Hence double-sided pneumothorax without any fluid was present. There were in both lungs small foci of bronchial pneumonia and at both bases small areas of pulmonary infarction. One of these at the right base presented the perforation into the pleura, that of the left could not be found. The diphtheria extended to the small bronchi.

The writer comments on the unusual location of the perforation. He considers that the rupture occurred while the child was asleep after its recovery from the first attack of dyspnœa, and that the cause was the pulmonary hemorrhage, which he discusses.

Case 2.—A child, 2 yrs. old, with tracheal and laryngeal diphtheria, rendering tracheotomy necessary. Immediate improvement followed, but that night the condition again became serious and the child died of asphyxia the following morning without any cause discoverable for this condition.

At autopsy there was found emphysema of the anterior mediastinum, and partial collapse of the left lung. The situation of the perforation was suspected to be an atelectatic area at the posterior inferior border of the left lung, but the hole was not demonstrated.

Case 3.—A 2-year old child with diphtheria, necessitating tracheotomy, which did not relieve, and the child died. At the autopsy left-sided pneumothorax and a much compressed lung were found.

Distension and anæmia of the lung tissue in the peripheral and superior parts with stasis of blood in the central and inferior parts are the result of laryngeal stenosis, and result in apoplexy of the lung tissue and rupture of the pleura.

Schwalbe's case is next reviewed. A 6¾-year-old diphtheritic child six days after a successful tracheotomy developed dyspnœa, emphysema, and double pneumothorax. Both sides were punctured, the air allowed to escape and recovery followed (*Deut. med. Wchs.*, 1891, No. 21).

V. Torday (*Archiv f. Kinderheilk.*, Vol. VIII) reports the case of a 3-year-old child with diphtheria. On the 7th day symptoms of laryngeal stenosis developed. On the 8th day was found consolidation of the entire left lung and the base of the right. On the next day subcutaneous emphysema appeared on the right side between the spine and the scapula which during the next four days spread over the trunk, face, arms. This subsided and the case evidently recovered. Torday discusses the cause and cites the only similar case (*Sachse, Virchow A.*, Bd. 52, p. 148).

Cnopf now adds a case of his. A diphtheritic child, 2¼ yrs. old, was tracheotomized; emphysema soon began in the neck and spread over the trunk. The child died the following day. Left-sided pneumothorax, and emphysema of the anterior mediastinum were found.

Hence diphtheria must be reckoned among the etiological factors of

pneumothorax because it causes stasis of blood and apoplexy, and distension of the lung with rupture of the alveolæ.

253. Aufrecht. Die Heilung des Empyems. *Deut. Arch. f. klin. Med.*, Vol. 52, p. 13, 1894.

On page 13 the author proposes to answer the question, "How does the healing of an empyema take place and the pleural surfaces again come in contact?" Roser's and Weissgerber's explanations are first disposed of. He then calls attention to the fact that the lung, collapsed as a result of the operation, must expand if the diameter of the opening in the thorax wall is smaller than that of the bronchus supplying that lung, since the diaphragm and thorax wall functionate on the operated as well as on the good side. But the adhesion between the pleural surfaces is necessary and this is only possible when they can build a pure fibrinous exudate.

254. Scott. Pyopneumothorax. *Cleveland Med. Gazette*, Aug., 1895.

After reporting three cases of pyopneumothorax resulting from empyema and commenting on the prophylaxis and treatment, Scott concludes: The diagnosis of pyopneumothorax circumscripta is at times extremely difficult. All obscure cases should be explored with an aspirating needle. The prognosis is good. Cases which have discharged through the bronchus and are improving should be treated by the tonic and stimulant plan. Cases which are not improving should have a pleurotomy performed in such a manner as to secure thorough drainage of the cavity.

255. Osler. Is the Coin Sound Distinctive of Pneumothorax? *Montreal Med. J.*, Vol. XXIV, p. 969, 1895.

Dr. Osler reports the clinical history and autopsy of a case of chronic tuberculosis, with a large cavity of the upper right lobe with thickened adherent pleura, in which case during life the coin sound was heard with great intensity. Until this case the general idea, and his also, was that this sign was distinctive of pneumothorax.

256. Lucas. Two Cases of Traumatic Pneumothorax. *Brit. Med. Jour.*, April 13, 1895.

Case 1.—Pneumothorax without fractured rib. Recovery. Boy, 2 yrs. 9 mos. old. He had been run over. Right-sided pneumothorax was found. Discharged in twenty-six days as convalescent. The lung signs had disappeared.

Case 2.—Extensive fracture of ribs. Pneumothorax. Man 43 yrs. old. Died five days later. Autopsy. Right-sided pneumothorax.

Lucas thinks in Case 1 there was no fractured rib. He discusses his idea of the mechanics. "The wheel passing over the chest first obstructs the air tube supplying some part of the lung; then continuing its pressure onwards, forces the air in the distended part beyond to rupture some of the air cells on the surface, and so allows the air to escape into the pleural cavity."

257. West. A Case of Obstinate Pleuritic Effusion with Negative Pressure in the Pleura; Accidental Pneumothorax; Recovery; Remarks. *Lancet*, March 16, 1895.

A woman, 19 yrs. old, had a small pleuritic effusion on the left side. Paracentesis showed negative pressure of —4 in. H_2O, respiratory oscillations of ½ inch. No fluid was obtained and air entered pleura (an ordinary trocar with a long tube extending to the floor was used). Succussion splash and signs of pneumothorax were now obtained. Paracentesis was again performed, and positive pressure of 4½ inches H_2O, respiratory oscillations of 1½ in. found. 97 ounces clear serum obtained. Recovery was satisfactory and in 16 days the patient was discharged.

West comments on the rapid absorption of air in an already inflamed pleura.

258. Paviot. Cas de pneumothorax au cours d'une pneumonie chronique de nature pleurogène et à forme ulcéreuse. *Cong. franç. de méd.*, Par., 1895, I, p. 898.

This case of Charcot's "chronic ulcerative pneumonia" was of a man, 59 yrs. old, who one month before admission to the hospital had pleuritic symptoms. Twenty days after admission he developed a frank migratory pneumonia of the right side, developing during two weeks; irregular temperature continued for eleven days; pneumothorax; death ten days later; autopsy.

The tissue was carefully studied, the writer is confident it was chronic ulcerative pneumonia, with the very rare complication of pneumothorax.

259. Rodet and Nicolas. Recherches expérimentales sur le pneumothorax. *Congrès franç. de méd.*, I, 1895, page 894.

The conclusions of former work in connection with Pourrat were that air injected into the pleura of a dog caused but insignificant disturbances provided the amount is not excessive; if sterile the air is utterly harmless; recovery is rapid at first, slower later, complete in about one month (in a dog).

The chemistry of the process is now studied.

In even three minutes they determined an absorption of O and excretion of CO_2.

To see if this interchange of gas is due to the respiration of the surrounding tissues, gas was injected into the tissue. The process was found different; it was slower; CO_2 is less rapidly excreted and O more rapidly absorbed. Injection of pure CO_2 into the pleura rapidly resulted in a mixture of CO_2, O, and N, hence tissue respiration is not indicated. If CO_2 be injected subcutaneously similar results are obtained, but much more slowly. They think here the contact of the gas with the blood vessels explains all this change.

To decide the importance of osmosis the animal was made to respire pure O and the amount of O in the pleura was too little increased to assume the presence of much osmosis with the alveolar air.

They conclude that the interchange with the gases of the blood explains all. Osmosis with the alveolæ is only indirect, through the intervention of the blood. The gas in the blood and pleura tends to come to an equilibrium, and its constant absorption leading to recovery is to be explained since each component of the mixture is at a higher tension than in the blood.

260. **Galliard.** Une complication insolite du pyopneumothorax; la migration spontanée du pus à la règion lombaire. *Bull. et mém. de Soc. méd. de Hôp. de Paris*, 3 s., XII, 1895.

He cites Delotte (*Thesis*, Paris, 1884) and Tuffier (*Soc. de Biol.*, 1892) concerning the migration of the pus in empyema. His case is unique since the patient had pyopneumothorax.

The case was; pyopneumothorax, total, long-standing, tuberculous in origin, on the right side; spontaneous migration of the pus through the diaphragm and lumbar muscles: large subcutaneous abscess in the right lumbar region; puncture of this abscess, drainage of pus from the pleura. Death.

The patient had had the pyopneumothorax for several months. Thoracentesis was several times performed and antiseptic fluids injected. The pus was not fetid. He left the hospital. Seven months later he was readmitted with all the previous signs of pyopneumothorax and tumor in the right lumbar region, 11 x 8½ cm. in size, fluctuating. The fluid in the chest was diminishing. Two weeks later (July 19, 1894) the fluid could not be demonstrated in the chest, and air can be demonstrated in the tumor. Only palliative treatment was attempted. Sept. 15, 1894, the patient died.

At autopsy the pleura contained only air. The right lung was airless, and the fistula in its superior lobe. There exists a fistulous tract from the pleura through the insertion of the diaphragm into the lumbar abscess.

261. **Galliard.** Contribution au diagnostic des Pneumothorax partiels du coté gauche. *Bull. et. mém. de Soc. méd. de Paris*, 3s., XII, p. 172, 1895.

It is often hard to distinguish between local pneumothorax and very large tuberculous cavities. In both may be heard amphoric respiration, metallic tinkling, amphoric cough, the bruit d'airain, and even the succussion splash. To recognize total pneumothorax from total cavity formation there remains only, on the right side, the depression of the liver; on the left side, the dislocation of the heart. He cites cases in which these points gave the correct diagnosis. But in tuberculosis, total cavity formation and total pneumothorax are both rare. It is partial pneumothorax due to the adhesions which occurs for the most part.

He proceeds to report cases of partial pneumothorax.

Case 1.—Pyopneumothorax dorso-axillaire of the left side, in a case of tuberculosis; amphoric respiration; no succussion splash. Death. Autopsy.

Case 2.—Tuberculous subclavicular pneumothorax. No fluid. Dis-

placement of the heart. No amphoric respiration, no bruit d'airain. (In this latter case there is no certainty of the diagnosis. The patient did not die.)

262. Meunier. CONTRIBUTION À L'ÉTUDE DU PNEUMOTHORAX: PROCÉDÉS CLINIQUES POUR MESURER LA TENSION DES GAZ ET LEUR TENEUR EN ACIDE CARBONIQUE. *Archiv gén. de Méd.*, 1895, Vol. I, p. 551.

Meunier first describes his method of study.

1. *The pressure of the gas.*—A simple water manometer is used attached to a Potain cannula. The proximal arm of the U manometer has a balloon dilatation of relatively large size which is half filled with the water. He warns to open the valve cautiously, and to close it if the patient begins to cough, also to avoid proximity of a lamp or other source of heat which can change the temperature of the air in the balloon.

2. *Analysis of gas.*—He recommends as sufficiently accurate the following method: The needle of a 20 cc. syringe is plunged into the chest and the syringe filled with the gas. The gas is then slowly forced out under an inverted eudiometer full of concentrated KOH. The CO_2 is absorbed and the remaining gas read. He thinks that the error is only from 0.5 to 1 per cent.

He then reviews previous observations on pressures and gases, and then reports three cases, one of closed pneumothorax, one of valvular and one of open, in which these points were determined.

CASE 1.—Closed pneumothorax. An acute pleurisy which became a pneumothorax through a mistake made in tapping. Ten days later he found:

Pressure on insp., — 8 cm. H_2O.
Pressure on exp., — 2 cm. H_2O.
Mean pressure, — 5 cm. H_2O.
CO_2 = 12 per cent.

CASE 2.—Valvular pneumothorax.
Insp. pres., + 7 cm. H_2O.
Exp. pres., + 9 cm. H_2O.
Mean pres., + 8 cm. H_2O.
CO_2, 9 per cent.
During recovery after the case had become a closed one:
Insp. pres., — 4 cm.
Exp. pres., + 2 cm.
Mean pres., — 1 cm.

CASE 3.—Open pneumothorax.
Insp. pres., — 1 cm.
Exp. pres., + 1 cm.
CO_2, 1.5 per cent.

Three rabbits were then experimented on.

Air was introduced in two cases until the pressure became 0, and in the third till it was positive. The pressure was then examined every day for 8 to 11 days. In the first two cases (starting with a pressure of 0) the

absorption of gas was progressive and regular, and the time necessary for the complete absorption of 80 to 90 cc. of air was 6 to 7 days. In the case in which the initial pressure was positive (after injecting 120 cc. of air) the absorption was rapid until the pressure reached 0 and then progressed as in the above rabbits.

At this point we wish to call attention to two points in Meunier's work. He evidently considered that the intrapleural pressure was an index of the amount of gas remaining in the chest; also that it was 6 to 7 days before he found normal pressures.

263. Levy. Über den Pneumothorax ohne Perforation. *Arch. f. exp. Path. u. Pharm.*, Vol. 35, p. 335, 1895.

Laennec first recognized the two varieties of this disease; symptomatic due to perforation, and essential. The latter was denied by others. Biermer and Senator expressed the belief that microorganisms could produce gas in the pleural cavity from the exudate, also Oppolzer, but more recently it was denied by many. Levy considers the following case proves its possibility.

The patient was a man, 48 yrs. old, who, following a cough of a few days' duration, developed a large left-sided pleural exudate. August 17, 1894, 2 liters of clear fluid were removed. Until Nov. 2 he was tapped five times. Nov. 8, signs of pneumothorax supervened (percussion note loud, tympanitic, metallic, somewhat amphoric. The Stäbchenpercussion gave a metallic ring. The heart was somewhat displaced, dulness very movable, no succussion splash.) Nov. 16 the chest was punctured and a slightly cloudy fluid obtained. Nov. 20, 200 cc. aspirated.

Nov. 24, operation for empyema. The seventh rib was resected. About two liters of opaque fluid removed.

Died Dec. 7.

Autopsy.—V. Recklinghausen. The case was found tuberculous; a small caseous nodule was present in the left lung, but no perforation of the pleura could be found. The fluid had been twice examined bacteriologically with especial reference to anærobic organisms and each time an anærobic gas-producing bacillus was found which he considers the Frænkel organism, and the infection he considers secondary to a serous tuberculous pleurisy.

264. Fütterer. A New Method of Treating Pneumothorax. *Medicine*, June, 1896.

The patient was a man, 27 yrs. of age, with right-sided pneumothorax. The method of treatment consisted in inserting a cannula in the highest part of the chest, through which the air could escape while the chest was filled through a lower cannula with a 0.75 per cent solution of oil of cloves and water. This was aspirated from time to time. In this way the author tried to remove all the air first and later, by aspiration and absorption, the fluid. Two months later the case had much improved.

265. Goodhard. Recurrent Pneumothorax. *Clin. Soc. Rep.*, London, 1896.

The interest in this case lies in the fact that the spontaneous pneumothorax affected first the right side and later the left.

The patient was a young man, 24 yrs. old, who had had a slight attack of pleurisy five years before, but until one week before the present illness had felt well. At that time he felt flying pains about the chest. The attack followed running up stairs. The symptoms and signs were, dyspnœa, hyperresonance, no true respiratory murmur on the right side, pronounced metallic tinkling. In two months he was well.

One year later he had a left-sided pneumothorax. For two or three months he had felt flying pains about the shoulders and chest, but nothing definite. For two or three days before the present illness after jumping up suddenly he felt puffy. The left side was drummy, especially at the apex. The cardiac impulse was in the epigastrium to the right of the sternum. There was well-marked metallic tinkling, the bruit d'airain was not obtained.

In neither attack had he had real distress, but considered himself well. The points of interest are: The recurrence on the other side, the absence of adequate cause, the rapid absorption of air each time, and the absence of pleurisy.

The rupture of an emphysematous bleb was considered the cause (of course the former attack of pleurisy suggests the possibility of tuberculosis as the underlying etiological factor).

266. Unverricht. Zur Behandlung des tuberkulösen Pneumothorax. *Verhandl. des. Congr. f. innere Med.*, Vol. XIV, 1896.

The surgical cases generally heal, the tuberculous generally die, but the reason for this difference is not clear. The workers in his laboratory had shown that a dog can absorb the most of the air introduced into the pleura in twenty-four hours, the oxygen disappearing first and the nitrogen last. He also tested the absorbing power of the inflamed pleura. In tuberculosis it is not probable that the absorbing power of the pleura is lost; the air must therefore be renewed.

He advises to open the chest in all cases of pneumothorax acutissimus and reports the good effect on two tuberculous cases. He advises free incision in such a manner that the air can enter and leave freely on respiration.

267. Lorrain. Méchanisme du pneumothorax à soupape. *Compt. Rend. Hebd. de la Soc. de Biol.*, 1896, p. 386.

As a result of the study of the cases seen in ten years the writer is certain that in this form of pneumothorax the air enters during expiration and not during inspiration, because in the two cases studied the pressure was positive during inspiration.

Case 1.—Insp., + 1 cm. H_2O; exp., + 6 cm.

Case 2.—Insp., + 5 cm.; exp., + 9 cm.

268. Unverricht. Zur operativen Behandlung des tuberkulösen Pneumothorax. *Deut. med. Wochenschr.*, May 28, 1896.

He reviews the various views on the subject. The name pneumothorax acutissimus is proposed for those cases which die on the first day after the onset, despite puncture. He considers that to this class belong most cases whose lungs are not the seat of advanced tuberculosis. Such cases should not be allowed to die, but be treated by incision or permanent cannula. In many cases of tuberculosis, however, it is better not to operate, since one may increase the pus secretion. Yet incision is indicated if there is any chance of the lung expanding again, even though the disease be advanced. The incision should be free enough that air can easily enter during the inspiration.

269. V. Weismayr. Ein Beitrag zum Verlauf des tuberculösen Pneumothorax. *Wien. klin. Wochenschr.*, No. 23, p. 485, 1896.

This is the case of a woman, 21 yrs. old, whose pneumothorax persisted five weeks less than five years, all of this time being pyopneumothorax (left side). The previous symptoms of tuberculosis were slight. During the latter months, at least, the fistula was open.

This remarkable case is minutely reported.

270. White (W. Hall). Two Cases of Pyopneumothorax in the course of Typhoid Fever, and Both Due to Straining at Stool. *Trans. of the Clin. Soc. of London*, Vol. XXIX, p. 105, 1896.

Case 1.—Man 19 yrs. old. On the 34th day of a very severe attack of typhoid fever with relapse, fluid was suspected at the left base. On the 41st day an enema was given. The patient strained a little, he suddenly became collapsed and livid. He then obviously had hydropneumothorax on the left side. The chest was aspirated twice. He died suddenly on the 44th day.

Case 2 was a boy, 12 yrs. old, with an ordinary attack of typhoid fever. Three days before temperature became normal (and about the 27th day of the disease), a pleuritic rub was heard at the left base, which disappeared on the following day. An empyema with fever developed, which was opened nine days after above-mentioned rub was heard. The following day, while straining at stool, pneumothorax suddenly developed, "and it appeared clear that a localized empyema, which already communicated with a bronchial tube (he had previously coughed up pus) had burst into the pleural cavity and that the boy had a pneumothorax." The chest was at once aspirated and twelve ounces of pus obtained. The boy recovered.

271. Aron. Experimental Studies of Pneumothorax. *Virchow's Archiv*, Vol. 145, p. 562, 1896.

These animal experiments are again reported in his publication of 1902 to the abstract of which the reader is referred.

272. Aron. Concerning the Treatment of Pneumothorax. *Deutsche méd. Wochensch.*, Sept. 3, 1896.

This is a case of pneumothorax with recovery, following the inhalation of hydrochloric acid fumes. There was no effusion, the gas was absorbed in about four weeks. This case is reported again in his monograph of 1902, to the abstract of which the reader is referred.

273. Gibson. Pneumothorax Produced by Physical Exertion. *Edinburgh Hospital Rep.*, Vol. IV, p. 237, 1896.

The patient was exercising with dumb-bells (of 8 lbs. weight), when he felt something give way in his chest and was attacked by dyspnœa. The pneumothorax seemed limited to the right postero-lateral region of the chest, extending from the angle of the scapula downward. Since localized he considers the cause the tearing of pleural adhesions by the exertion.

Other cases of pneumothorax due to exertion are mentioned.

The patient was well in one fortnight. There was no evidence of fluid in the chest.

274. Chauffard. Du pneumothorax simple et de son diagnostic par la tuberculine. *La Semaine Médicale*, Vol. XVI, p. 501, 1896.

The patient, a woman 30 yrs. old, had a left-sided pneumothorax following a violent movement of the left arm while laughing (she picked up her child); violent pain and dyspnœa followed. She was admitted on the fifth day of the disease. No fluid was present. The whole chest was tympanitic, with absence of respiratory murmur and vocal fremitus. No metallic phenomena, no tinkling, no succussion splash were present, but a splendid coin sound was obtained. The chest was not distended, the heart not displaced. The temperature was normal. In this case for the first time the tuberculin reaction was used to exclude tuberculosis. It was negative. The writer emphasizes the value of the test.

275. De Bovis. Des indications et des résultats du traitement chirurgical du pneumothorax non traumatique. *Gaz. des Hôp.*, Vol. 69, p. 717, 1896.

The conclusions of this article are:

1. The treatment of pneumothorax depends essentially on its cause and form.

2. The operative indications proceed from the asphyxia and pleural complications.

3. The asphyxia is usually amenable to thoracentesis. It may not always be innocuous (in a tuberculous case). It is here an indication of urgency. In the emphysematous it results in cure.

4. Pleural complications demanding intervention are the presence of pus or gangrene. Pleurotomy should be precocious in this latter case. In tuberculous it may be sometimes delayed to allow the formation of adhe-

sions. One hastens more or less according to the more or less marked presence of a pyemic state. It gives in general good results.

5. The resulting thoracic fistulæ may be drained if the general condition allow to drain at the same time the visceral fistula.

The discussion of the subject is long and evidently thorough, and is based on literature rather than personal cases. Over fifty references are given.

276. West. Intrapleural Tension. *Allbutt's System of Medicine*, 1899.

The forces of which the intrapleural tension is the resultant are:

1. The rigidity of the chest wall, which may be disregarded in the adult, but not in a child.

2. The elasticity of the lung.

3. Movements of respiration.

The respiratory oscillation in the bronchi is in inspiration —0.5 mm. Hg.; in expiration, + 2 to 3 mm.

The intrapleural tension after death is equal to the atmospheric pressure minus the elasticity of the lung; during life to this minus 0.5 mm. Hg. in inspiration, plus 3 mm. at expiration. The pressure by violent expiration may be raised to 70-100 mm. Hg.

The elasticity of the lung (quoting Donders) is 6 to 8 mm. Hg.

Since the mediastinum is movable, to open one chest satisfies the elasticity of one lung completely and much of the other. If in pneumothorax the lungs are adherent the change in respiration will be less, since there is less elasticity exerted; hence the results of pneumothorax are more severe in a healthy chest than in a diseased one.

If the pleura over one lung be adherent the other lung enlarges. This so-called compensatory emphysema is in reality compensatory hypertrophy, since the elasticity is also increased.

The contractility of the lung is due to its elasticity and also to its muscle fibers, which give the *pulmonary tone*. This pulmonary tone is diminished in pneumonia, typhoid fever, delirium tremens, erysipelas, and phosphorus poisoning (quoting Perls), where the lung is not involved locally, but presents a loss of neuro-muscular tone, the result of general neuro-muscular failure, or of local nutritional disturbance; it is similar to abdominal tympanites. This loss of pulmonary tone is indicated by its Skodaic resonance on percussion. "I have on several occasions seen this set in without any local disease of the lung"—"acute pulmonary tympanites." Where there is local disease the other parts of the lung frequently yield this tympanitic tone, e. g., over a pleural effusion, and just above a pneumonic area. In the latter case since there is neither overdistension nor collapse of the pulmonary tissue, West attributes it to "loss of tone of neuro-paralytic origin due to nutritional disturbance."

The Pleural Cavity under Pathological Conditions.

Pneumothorax air may enter:

A. Through the chest wall.

1. In punctured wounds. In this case the air usually crosses the pleura to the subcutaneous tissue, causing emphysema, rather than pneumothorax.

2. By a small opening through which the air enters more easily than it leaves. The conditions are about the same as in pulmonary wounds.

3. By an opening as large in cross section as the trachea. Here its elasticity tends to collapse the lung, but since the intrabronchial tension in expiration is + 1½ to 2 mm., it will not entirely collapse, probably not more than one-half. This is well seen in cases of empyema immediately after operation, in which case the lung expands when there is no violent expiratory movement. West mentions two cases of his of 18 and 5 months' standing. Immediately after operation the lungs filled the pleural cavity.

B. Air from the lung.

1. Theoretically there could be a hole in the lung large enough to allow the air to pass in and out freely, but this is hardly ever seen.

2. Air entering the cavity more easily than it leaves, hence the pressure rises during expiration, compressing the lung; but "although . . . the mediastinum may be displaced to a maximum and the lungs be completely collapsed in cases where there is no expiratory compression, still in the great majority of cases this rise of pressure during expiration plays a very important part in the production of both these phenomena."

West does not think the classification open, closed and valvular forms, very important; in the early stages it is always more or less valvular, and when the lung is completely collapsed the opening is always closed.

Inspiration can raise the pressure above that of the atmosphere only in dyspnœa, and then not much; hence if in ordinary pneumothorax the inspiratory pressure be positive some other factors (usually fluid) are to blame.

The expiratory pressure is always positive.

The respiratory oscillations vary with the violence of the respiratory movements, but even when this is great the oscillations need not be much since so much of the elasticity of both lungs is satisfied.

West now summarizes 20 observations on intrapleural tension in cases of pneumothorax. In two of these the inspiratory pressure was 0 and in both fluid was present as well as air. "From these it is evident that as soon as the fluid forms air must be absorbed, since the opening into the lung was in both cases closed."

The inspiratory pressure in the others varied fom 0 to + 9 in. "It is not a matter of wonder that the inspiratory pressure rises as the fluid forms, but it is surprising that the pressure is not higher than we find it. The highest pressure that I have observed was 9 in. H_2O, but pressures as high or even higher have been met with in serous effusions. It follows, therefore, that when fluid forms in pneumothorax a large amount of the air present must be absorbed as the fluid forms." It is well proven that the pleura can absorb air, hence "I think we may possibly go so far as to say that if the intrapleural pressure remains unusually high in pneumothorax it may be taken as an indication that there is extensive disease both in the lungs and pleura, so that the absorption of air which would ordinarily occur is prevented from taking place."

The expiratory pressure ranged from 0 to + 13¼ in. H_2O.

The respiratory oscillation ranged from 0 to 8 in.

"These observations show that in pneumothorax whatever general statements may be made they have to be applied with caution in individual cases, for it is impossible in any given case to forecast what the actual pressures will prove to be."

277. Lesné. Formes Cliniques du Pneumothorax. *Gaz. des hop.*, Paris, Vol. 69, p. 774, 1896.

This is a discussion of the subject based on literature without the report of cases or of original work. The references in literature are not given. It is an excellent essay on the subject.

278. Galliard. Le pyo-pneumothorax de nécessité. *La médicine moderne*, VII, p. 113, 1896.

By this term he means the tumor in the thoracic wall, caused by the migration of pus and air from the pleura.

After reviewing the literature of the subject and discussing it he concludes by giving the clinical picture of the condition.

The patient with pyopneumothorax will have, after severe pain, the appearance of a tumor in the region of the nipple. The tumor is soft, fluctuating, crepitant, is reducible, is increased by coughing or efforts, and by the movements of the patient. This tumor can open externally, discharging pus and gas, or migrate to the lumbar region or anterior abdominal wall. One complication is general or local subcutaneous emphysema. The diagnosis is easy, the prognosis grave, and the treatment operative.

279. Sackur. Zur Lehre vom Pneumothorax. *Zeitsch. f. klin. Med.*, Bd. 29, p. 25, 1896.

In a brief review of previous work, in which that of Leichtenstern, Guttmann, Weil, Rodet and Pourrat, Krebs, and Blumenthal are discussed, Sackur criticises Weil's since he measured the excursion of the chest wall, and Krebs since his animals were narcotized, hence the respiratory center's irritability was much reduced. Since Blumenthal had shown the effect of the difference in size of the cannula used to produce the pneumothorax, Sackur conducts his experiments as follows: He will make a hole in the chest too large to admit of respiration on that side; he will measure the air respired, and work on non-narcotized rabbits. The animal was tracheotomized, the air of inspiration and expiration separated. The air used per 1 min. and the rate of respiration were measured.

Result 1. Immediately after the production of an open pneumothorax, and also later, the amount of air used per 1 min. is just above normal (e. g., normal amount per 1 min., 375 cc.; in 30 min. after pneumothorax formed, 434 cc.; in 2½ to 5 hours later, 305 cc.; in 6 to 8 hours, 390 cc. On another rabbit these figures were: before, 410 cc.; after, at same intervals, 450 to 480 and 400 cc.). Hence the one lung uses as much air per 1 min. as both lungs normally, since (1) the type of respiration changes; (2) the

rate increases. The rate is often higher immediately after the operation than later. The depth (amount of air breathed per 1 respiration) is, of course, less.

Result 2. The absolute amount of air used in each respiration is less than normal, but more than one-half normal, hence the one lung breathes deeper than normally.

By what mechanism does the animal make one lung use as much air as normally do both?

Result 3. If the vagus nerve of the pneumothorax side be cut, no change results. If both vagi be cut the amount of air respired per one minute does not change, but compensation is effected by increased depth of respiration, rather than increased rate. Hence the mechanism is not nervous.

Result 4. The arterial blood pressure in the carotid does not change after the chest has been opened, although there is now no negative pressure in the thorax to aspirate the venous blood, hence changes in the amount of blood are not the mechanism controlling the changes in respiration, even when both vagi are cut. Hence it must be qualitative changes in the blood.

Result 5. The oxygen in the blood drops about one-half after the thorax is first opened. (The Geppert method of gas analysis was used.) This is in accord with the dyspnœa of animals in reduced atmosphere (Sackur reports some of Geppert's experiments). In right-sided pneumothorax the animals, as a rule, died, since in rabbits this lung is about one-third larger than the left.

Result 6. The CO_2 showed no constant changes. Conclusion—changes in respiration after opening the chest are due to changes in the O-content of the blood.

In open pneumothorax, if the opening be closed, a dying animal may be restored, and the O in the blood return to normal, since then the lung can take some share in respiration. (He thinks these results throw light on human pathology, but only mentions the subject.)

Protocols of twenty-three experiments follow.

280. Rodet and Nicolas. SUR LE PNEUMOTHORAX EXPÉRIMENTAL. DES MODIFICATIONS SUBIES PAR UNE MASSE GAZEUSE INJECTÉE DANS LA PLÈVRE. *Arch. d. phys. norm. et path.*, 5 s., VIII, p. 640, 1896.

Historical.—Mention is made of the work of Davy (1824), Brière de Boismont (1825), Martin-Solon (1865), Apjohn, Ulm, Pepy.

The work of Demarquay and Lecomte is reported at length; also Ewald's, Hoppe-Seyler's, Wintrich's. Potain injected sterile air as a therapeutic measure and found that this artificial pneumothorax was completely absorbed in about one month. He also experimented on animals.

Technic now is described. A very fine glass cannula was used to inject the gas, and it was withdrawn through the same cannula. The technic is most careful. KOH and pyrogallic acids are used to determine the CO_2 and the O.

I. First series showed (1) that air introduced into the pleural cavities of dogs is gradually absorbed, at first rapidly, then slowly, and is gone in a few weeks. (2) The gas in the pleura after the second day contains considerable CO_2 (7 to 15 per cent) and less O (12.9 to 18.9 per cent).

II. The changes in the first few hours and minutes are now studied.

The composition of air injected into a dog, in

	5 min.	15 min.	3 min.
CO_2	3.4 per cent	3.2 per cent	2.9 per cent
O	15.0	18.8	18.1
N	81.6	78.0	79.0

Hence the changes are rapid, the exhalation of CO_2 and the absorption of O.

III. The explanation is now sought. There are three possibilities.

IV. (1) The respiration of the pleural tissue. But the change is too rapid, and the O should progressively disappear, and the CO_2 increase. Also an experiment was made, pure CO_2 being injected, and after remaining various periods (10 to 22 min.), removed. O and N are at once exhaled, in 14 min., e. g., the composition being

CO_2	8.5 per cent
O	16.2
N	75.3

But in animals which die soon the interchange of gases is much less, showing the importance of the respiratory movements. Hence tissue respiration cannot explain it.

V. Osmosis is now discussed, but the pleura is a thick wall between pleura and alveolæ.

Into the pleura of a dog breathing pure O is injected air, and in four minutes this is removed.

CO_2	4.4 per cent
O	21.8
N	73.8

This they think rules out osmosis between the pleura and the alveolæ, full of pure O.

VI. Interchange between the pleura and the gases of the blood, in the sub-pleural vessels, is the only remaining possibility, although the tissue respiration is a factor.

281. Galliard. Statistique du pneumothorax dans les hôpitaux de France. *Bull. et. mem. de la Soc. méd. de Hôp. de Paris*, 3 s., XIII, p. 253, 1896.

After reviewing the work on the subject Galliard calls attention to the two questions before the statistician.

(1) Of each 100 living tuberculous patients how many have pneumothorax?

(2) How many fatal cases of phthisis die of pneumothorax?

In conclusion there were 2253 male phthisical patients, of whom 25 had pneumothorax, or 1.11 per cent; 1162 women, of whom 11, or .946 per cent had pneumothorax.

That is, on January 22, 1896, at 10 a. m., of 3415 phthisical patients in the hospitals of France, there were 36, or 1.054 per cent, cases of pneumothorax.

The classification according to sex, age, side affected, effusion, etc., is quite minute.

282. Flory. A Case of Pneumothorax. *Lancet*, Jan. 9, 1897.

Evidently pneumothorax (a man of 50 years), of tuberculous origin, with a sudden onset.

283. Tuffier and Hallion. *Lancet*, Feb. 20, 1897.

At a meeting of the surgical society of the Academy of Medicine, of Paris, these men proposed in lung surgery to avoid collapse of the lung by a tube, put in the trachea, surrounded by a rubber bag, which by inflation would plug the larynx, and through the tube the necessary pressure could be maintained.

M. Delorme replied that if the patient coughed he would accomplish the same.

284. Lavernhe. Contribution to the Study of Pneumothorax. Lyons, 1897.

The history, prognosis, treatment, and occurrence in Algiers are studied. Nineteen cases are referred to briefly, and seven reported at length.

Conclusions—(1) In Algiers pneumothorax is of more common occurrence in tuberculosis than in France. (2), (3) In cases difficult of diagnosis the tuberculin test should be tried. (4) In simple pneumothorax perform paracentesis, if the intrapleural tension rises and gives rise to symptoms. (5) In hydropneumothorax of non-tuberculous origin follow the expectant method of treatment, also if tuberculous. (6) In non-tuberculous pyopneumothorax early operation gives remarkable results. If tuberculous operate at once, free incision being made if the patient is in good condition, small if bad. (7) The attempts made by the pulmonary surgeon to obtain closure of the fistula, although few are encouraging. (8) Radioscopy is of use in following the case.

285. Sackur. Weiteres zur Lehre vom Pneumothorax. *Virchow Arch.*, Bd. 150, p. 151, 1897.

This is an answer to Aron's criticism of Sackur's work. They both found slight changes in the blood pressure when a pneumothorax was made, but Sackur considered them too small to notice. Aron, on the other hand, considers them important.

Sackur adds a computation of the circulation through the lungs and decides that in pneumothorax more blood flows through the collapsed, not respiring lung, than through the other.

286. **Kidd.** Pneumothorax Caused by Direct Violence, Without Fracture of the Ribs. *Lancet*, 1897, II, p. 1243.

The patient, a man 21 years old, fell from an omnibus, striking on the left side of his back. Pneumothorax followed, and later a liquid effusion and fever. There was no external wound, no fractured rib, no hæmoptysis. Complete recovery in three and a half months.

Kidd considered it a case of rupture of the lung in a healthy person, (but the effusion and fever, and the long time before recovery, suggest the possibility of preexisting disease of this lung).

287. **West.** The Prognosis of Pneumothorax With Some Statistics as to Mortality and Duration, and an Account of a Series of Cases of Recovery. *Lancet*, 1897, I, p. 1264.

The *mortality* of pneumothorax is highest in the first few days after the onset. The general mortality being about 70 per cent. Of 39 cases, 10 died on the first day, 18 during the first week. Of 74 cases, 45 died in the first month. One was alive at the end of nine months.

Death is due (1) to the pneumothorax itself, or (2) to the complications of that trouble, empyema for illustration, or (3) from the original disease, e. g., phthisis.

In death due to the pneumothorax the cause may be suffocation due to the suddenness of the change, not to the amount; hence if the person can be kept alive a few hours, until circulation and respiration readjust themselves, the life may be saved; or to suffocation from the aspiration into the other lung of the contents of a tuberculous cavity; or to shock, which is rare, e. g., Lebert's case.

If death is due to complications, it is often the result of the *effusion* consequent on the pneumothorax. The effusion is serous in one-third of cases, seropurulent in one-third, and purulent in the remaining third. In the latter case operation is necessary, the case must be made one of open pneumothorax, or the pus will point and it become so by rupture.

The *prognosis* varies as the amount of dyspnœa which is relieved by paracentesis, and the condition of the other lung. This lung is overworked and if it gives way bronchitis will develop, hence bronchial râles are a bad omen. The effect on the heart, the general strength of the patient, and lastly the nature of the underlying disease also are important elements of the prognosis. The most favorable cases are the traumatic, the least the gangrenous.

Recovery is complete in not more than ten per cent. The most favorable are those with no effusion.

Then follows an interesting series of eleven cases of recovery, which he reports.

Cases of Recovery Without Effusion of Fluid.

Case I.—Acute phthisis, pneumothorax of the left side developing in the hospital; complete recovery in five weeks; rapid progress in the phthisis, and death subsequently at home from phthisis.

Case II.—Pneumothorax of the left side, without definite symptoms at the time of onset, without effusion and with complete recovery.

Case III.—A man under treatment for possible phthisis who developed right pneumothorax; complete recovery in six weeks; well twelve months afterwards.

Case IV.—Pneumothorax of the right side; probably the third attack, no fluid; complete recovery.

Case V.—Pneumothorax in an apparently healthy girl; no effusion; complete recovery. Page 1331.

Cases of Recovery After Serous or Seropurulent Effusion.

Case VI.—Pneumothorax with seropurulent effusion of three months' duration.

Case VII.—Case of phthisis under tuberculin treatment; left pneumothorax; serous effusion; paracentesis; recovery; recurrence three weeks later without effusion; recovery.

Case VIII.—Pneumothorax of the left side in an apparently healthy person; without marked symptoms considerable effusion spontaneously absorbed; complete recovery; six months later pneumothorax of the left side with much dyspnœa, without effusion, also with complete recovery.

Case IX.—Pneumothorax with serous effusion; paracentesis; absorption of air; partial recovery.

Cases of Recovery With Purulent Effusion.

Case X.—Left pneumothorax; serous effusion; paracentesis; suppuration of puncture; empyema; sinus left; death from phthisis some months later.

Case XI.—Left pyopneumothorax; discharge through the lung; partial recovery.

The Discussion of this Paper. *Lancet*, Jan. 16, 1897.

Sir **Dyce Duckworth** pronounced the paper the most complete in several years, and he "thought the presence of pus in the pleural cavity less harmful than might be expected."

Kingston Fowler thought it best to let cases of pyopneumothorax alone. West joined issue with these men and urged operation for pyopneumothorax.

In this discussion **G. R. Turner** mentioned a case of pneumothorax from rupture of the œsophagus in an attack of vomiting.

288. West. Pyopneumothorax of Several Months' Duration Cured by Free Incision; With Some Remarks on the Surgical Treatment of Pneumothorax. *Brit. Med. Jour.*, 1897, II, p. 1568.

West describes such a case at length. He then calls attention to the fact that in this case the lung had been collapsed for some time, yet without the formation of adhesions. In one case the lung expanded after eighteen months' collapse. He next vigorously combats the idea that collapse of the lung favors the healing of tuberculosis. They formerly claimed this also for pleurisy.

A discussion of the surgical treatment of various stages of the disease follows.

289. **Ewart.** A Case of Acute Pulmonary Gangrene in a Healthy Youth; Pyopneumothorax With Severe Symptoms; Incision and Drainage; Recovery. *Lancet*, June 19, 1897.

Laennec, in 1823, first described gangrene of the lung. Ewart then speaks of the causes of this disease, giving several references to the literature.

A young man, 18 years of age, of good past history, had an acute pulmonary attack diagnosed as "diffuse bronchopneumonia, probably post-influenzal in origin." The breath and sputum became foul smelling on the eighth day. On the nineteenth day of the disease empyema having developed, aspiration was decided upon, but while preparing him for the operation pneumothorax was discovered. Two days later the diagnosis of pneumothorax due to gangrene of the lung being decided upon, operation was performed, and much fetid pus removed. About six weeks later he was almost well.

Ewart calls particular attention to the absence of any clue to the source of the infection; to the sequence in this case of bronchopneumonia, empyema, bronchiectasis, and gangrene with pneumothorax; to his desperate condition, and recovery after the operation.

290. **Khinitz.** The Value of Puncture in the Treatment of Pneumothorax. Paris, 1897.

After a short review of the subject and especially of the history of paracentesis, all the views advanced receiving due consideration, the author reports one case, quotes several others thus treated, and arrives at the following conclusions:

1. Puncture is a method which should not be neglected.
2. It gives the patient instant relief.
3. It is especially indicated in valvular pneumothorax.
4. In valvular pneumothorax he recommends a permanent cannula, as preferable to repeated tappings.
5. Tapping is not justified in pyopneumothorax except as a palliative measure to relieve a hopeless case. In all other cases free incision and drainage are indicated.
6. The progress of pulmonary surgery justifies trials which may give good results.

291. **Galliard and Bruneau.** Fatal Pneumothorax in a Case of Extreme Emphysema. *Jour. des Praticiens*, 1897, p. 287.

A man, 72 years old, atheromatous and emphysematous, as a result of catching cold developed bronchitis and pneumothorax of the right side. He died the next day.

292. Reineboth. (Experimental Study of the Redistension of the Lung When the Thorax is Opened, Especially After the Operation for Empyema.) *Deuts. Archiv f. klin. Med.*, Vol. 58, March, 1897.

The writer seeks an explanation to the undeniable fact that with the chest open, after the empyema operation e. g., the lung will again expand filling the pleural cavity. He mentions the theory of

Roser (*Berl. klin. Wochensch.*, No. 46, 1878), that the lung becomes progressively adherent to the chest wall, starting at the hilus. The adhesions are later absorbed, setting the lung free; of

Weissgerber (*Berl. klin. Wochensch.*, 1879, No. 8, S. 107), who thinks that the active factor is the positive pressure on expiration in the bronchi, and especially on coughing, which distends the lung, since the expiratory variations in pressure are greater than the negative on inspiration; hence little by little the lung is inflated; of

Bouveret (*Traité de l'empyème*, 1888), who in those cases taking a long time to heal, agrees with Roser, but for those requiring but a few days with Weissgerber in part, but thinks that by adhesions the lung holds the expansion it has already accomplished. He advises the use of a bandage with valvular action.

Schede (*IX Congr. f. inn. Med.*) called attention to the collapse of the lung in inspiration, and its expansion on expiration, so that with the chest wide open the lung may even protrude from the wound, and rejected Roser's idea since the pleura cannot produce granulation tissue.

Gerhardt (*Deutsche Chirurgie*, XLIII, 1892) believed that the lung was distended by expiratory movements and held in place by adhesions.

Aufrecht (*Deuts. Arch. f. klin. Med.*, 1893) rejected Roser's idea, and said that the descent of the lung on inspiration can be detected clinically, and denied that ordinary respiration could distend the lung. According to Aufrecht the collapsed lung is distended by each inspiration when the diameter of the external wound is smaller than that of the primary bronchus leading to that lung, but the lung collapses just as much on the following expiration, and only after the exudate of the receding pleurisy is almost purely fibrinous will the lung hold the gain it has made on inspiration.

Schede (*Handbuch der spec. Therapie inn. Krankheiten von Penzoldt und Stinzing*, 1895) agrees with Weissgerber and believes that fibrinous adhesions hold the gains made in expiration, and recommends a valved bandage.

I. *Respiratory movements of the affected lung in pneumothorax in general.*

Reineboth quotes Sehrwald concerning the importance of the size of the hole in the chest wall, and states the problem thus: The collapsed lung is a membrane attached to a tube (the bronchus) and facing on one side the pleural cavity, on the other the trachea and the sound lung. If therefore the respiratory movement cause equal changes of pressure in the pneumothorax cavity as in the interior of the collapsed

lung there will be no change in the lung, and this will be the case when there is a mathematical equality between the air entering in a unit of time through the external orifice and the bronchus of the *sound* lung. If, however, the external orifice is smaller than the bronchus, inspiration will reduce the pressure in the pneumothorax cavity and air will rush in through the bronchus to distend the lung, etc.

II. *The respiratory movements in open pneumothorax when the fistulous opening is smaller than the bronchus.*

In a rabbit by exposing the parietal pleura one can see at each systole the edge of the lung expand over the margin of the heart.

1. If now the pleura be opened by the finest hollow needle, the lung recedes along the thorax wall, returning a little with each inspiration, for about six breaths, then leaves the chest wall and collapses towards the hilus.

2. If a larger hole be made the collapse is quicker and the rhythmical expansion with inspiration is less marked.

3. If now the animal be turned so that the hole in the chest wall is lowest, and by shaking the lung brought to the chest wall, it can be seen to expand with inspiration and retract just as much with expiration.

4. Open the pleura widely till the lung is entirely collapsed. At the height of inspiration clamp the trachea and during the violent expiratory movements the lung expands.

5. A manometer is put in the pleural opening of a patient whose rib was resected in an empyema operation, also a catheter, and the pleural tension noted, with the hole of various sizes (made so by partly clamping the catheter).

Conclusions from the above:

Any hole in the pleura causes collapse of the lung, and the time required varies inversely with the size of the hole.

During the collapse the same changes in volume due to respiration are seen, and are more marked the smaller the hole, and continue if the opening was very fine after complete collapse.

III. *When the external hole is larger than the bronchus.*

Conclusions: In ordinary respiration there are no volume changes in the collapsed lung. Forcible inspiration causes further collapse, and expiration an inflation (the trachea being clamped).

IV. *Other opinions concerning the motions of the pneumothorax-lung.*

The above experiments have shown that the collapsed lung is inflated by the sound lung in forcible expiration, whatever the size of the opening in the chest wall, but the volume changes during respiration vary according to the relation between the size of this hole and that of the bronchus. If therefore the hole is large he agrees with Weissgerber, Bouveret and Schede that the lung expands in expiration and contracts in inspiration, but if relatively small the reverse is true.

Carlett and Strauss in an old empyema case found the lung expanded on inspiration and contracted on expiration, but their manometer made this a closed pneumothorax.

Aufrecht denies inflation by common expiration; it merely resumes the condition it had before the preceding inspiration, but Reineboth answers this by experiments on a suitable case, showing that in quiet respiration it took a force of 150 to 200 gms. to prevent this lateral motion of the lung during expiration. But the objection might be made that this lateral motion was transmitted from the mediastinum. Reineboth therefore quotes Sehrwald, showing that during the expiration the mediastinum slackened and was pushed a little towards the good side. In animals with a thinner mediastinum, on the other hand, it is aspirated to the good side in inspiration. He claimed the collapse on inspiration was only an apparent one, due to its flattening on being pulled down by the descent of the diaphragm.

V. *The influence of former adhesions and of the heart-beat on the motion of the lungs in open pneumothorax.*

If the opening is large, during inspiration the adherent part expands with the chest wall, the free part collapses. During expiration the adherent area is forced in, the free bulges. This was well seen in a suitable case he observed. The lung expands with each systole in open pneumothorax, therefore this expansion of the part of the lung against the heart should cause a depression of the free-lying surface, which he saw occur.

VI. *The influence of a bandage on the respiratory movements.*

The tube of a manometer was inserted into the operation wound of an empyema case, and the intrapleural tension measured. The effect of the bandage was equivalent to decreasing the size of the fistula.

VII. *The influence of coughing on the motion of the lung and on the intrapleural tension.*

In animals the cough was simulated by clamping the trachea at the height of inspiration. This is followed for forcible expiratory muscles which inflate the collapsed lung irrespective of the size of the hole. On his patient with an exposed part of his lung he could see the preliminary drawing in of the surface on the deep inspiration and the subsequent protrusion in the expiration of cough which lifted even 1000 gms. In forcible expiration also the mediastinum is pushed to the pneumothorax side.

VIII. *The value of adhesions in the healing of a pneumothorax.*

Roser's progressive adhesion theory is true in some chronic cases, as after Eslander's operation, but Weissgerber's criticism is good that such a process would unite surfaces not in contact when the lung is fully distended. Old adhesions (those formed before the collapse) are of aid in guiding the lung to the chest wall (e. g., Blazejewski, *Berl. klin. Wochensch.*, 1890, page 562). For adhesions to form requires a longer time than during coughing, and they follow the deposit of pure fibrin without many leucocytes.

The rôle of adhesions:

Schede said they are of secondary importance. Weissgerber said if formed before a certain expansion has occurred they are a hindrance.

But if the lungs are partially distended the adhesions maintain the gain made.

Examination of pleural cavities after the healing of an empyema shows some obliterated, some not. Clinically the edge of the lung is sometimes movable on inspiration, sometimes not.

IX. *Elasticity of the lung during healing.*

A loss of elasticity would be advantageous and is easily conceivable since empyema follows disease of the lungs and pleura, also the lung has been compressed and its circulation changed. The effect of intermittent expansion also is to diminish elasticity.

X. *The absorption of air in the pleuræ* is little at first until the pleura has become cleaned, then is perhaps the most important factor.

XI. *The forces in the distension of the lung in conclusion* then are, by repeated dilatation of the lung from forced expiration. Of more importance is the negative pressure produced in the pleural cavity by the forcing of air by coughing, etc. (aided by the valvular action of the bandage). Also the full breaths (inspiration) following coughing by increasing the negative tension, aid. In addition are important the adhesions, the absorption of air, and the loss of elasticity of the lung.

XII. *Obstacles to expansion* are, adhesions at hilus, thickened pleura, atelectasis of the lung, the condition of the other lung.

XIII. *Application of above in treatment.*

Most important is a good bandage. A small incision is no better than a large one. Forced expiratory efforts (pulmonary gymnastics) and removal of the fibrin coat also are useful.

The value of this paper is hard to estimate since he does not give us the evidence necessary to judge of the amount of experimental work upon which he bases his conclusions.

293. Comby. (Pyopneumothorax, Gangrenous, in a Child; Pleurotomy; Recovery.) *Jour. des Praticiens*, 1897, p. 302.

A healthy boy, 11 yrs. old, had a sudden pulmonary attack, with effusion at the left base, and three days later pneumothorax. On the 9th day 500 gms. of very fetid pus were aspirated, hence pleurotomy was done, the pleural cavity washed out with boric acid and drained. Subsequent lavages with 1:1000 $KMnO_4$ were used. Recovery in three weeks. Netter examined the pus, found several varities of organisms, but considered the streptotrix funiculi the agent causing the putrefaction.

294. Widal. (Putrid Pleurisy without Gangrene of the Lungs or Pleura. Pneumothorax from the Gaseous Exhalation.) *La Semaine médicale*, 1897.

A man, 45 yrs. old, died on the eleventh day from pyopneumothorax with very fetid exudate. No lesion of the lung could be found, no perforation of the pleura, no portal of entry of the infection.

In the fluid were found great variety of aerobic and some anaerobic

organisms. Animal inoculation gave a gaseous, gangrenous abscess and a pure culture of proteus vulgaris.

A somewhat similar case, but one in which the evidence of pneumothorax is not given, is reported by

294a. Courtois-Suffit. *La Semaine*, 1897, p. 460.

295. Béclère. TREATMENT OF PNEUMOTHORAX. *Jour. des Praticiens*, Sept. 4, 1897.

Béclère will speak only of pure pneumothorax following pulmonary disease. He mentions the causes of such, wound of the lungs, superficial tubercle, emphysematous bleb, etc., and then of the forms open, valvular, closed. In the valvular form air enters first in inspiration, later in forcible expiration. At the onset it is open or valvular, later closed. Valvular pneumothorax deserves the name "suffocative pneumothorax," because of the high tensions present which compress the lung and impede circulation.

At the onset the pain and dyspnœa are to be combated with morphia. Dry cups over the chest combat the congestion in the other lung. If this congestion is intense (as shown by the râles) he advises wet cups or venesection. The cardiac collapse is met with an ice bag over the heart, subcutaneous injections of ether, caffein and camphor, and the asphyxia by oxygen inhalations.

Aspiration is useless in the open form. If closed it is needless and dangerous, since it may cause rupture of the closed fistula; if valvular, it is unnecessary, since the superior intrapleural tension will cause the exit of air. Hence aspiration is never indicated. But puncture of the chest is indicated in the valvular form when the high tension is indicated by the extreme dyspnœa, the circulatory disturbances, the increased hyperresonance of the note, the accentuation of the amphoric quality of the bruit d'airain, and of the metallic tinkling, the maximum displacements of the neighboring organs. Do not hesitate to puncture; if useless it has done no harm. But use a needle of a hypodermic syringe, for the finest trocars are dangerous, causing subcutaneous emphysema.

To avoid inspiration of air through the needle he attaches a rubber tube to it about one-half of a meter long, at the other end of which is a glass tube about twenty centimeters long which is plunged vertically in a test tube of water, which serves as a manometer and allows one to see if any air escapes from the chest.

296. Merklen. (THE FEVER OF TUBERCULOUS PNEUMOTHORAX.) *La Presse Médicale*, May 29, 1897.

A detailed history of one case is given, and the temperature chart. Among the interesting points in the case was the sharp rise of temperature at the onset of the pneumothorax and its persistence during the following two months, due, he says, to the extension of the infection to the pleura.

Pneumothorax alone does not cause fever, as is seen in the cases following emphysema, but here we have pneumothorax plus pleurisy, and the sudden rise in temperature marks the new infection by the tubercle bacillus and is the same as the tuberculin reaction produced by its injection. The temperature was very irregular. Aspiration gave serous not purulent fluid, as the temperature would have led one to expect. The temperature has little prognostic value, it indicates here only tuberculous pleurisy.

297. West. Treatment of Pyopneumothorax. *Lancet*, Nov. 27, 1897.

A man, 34 yrs. old, with left-sided pneumothorax and seropurulent effusion was operated on, since he was getting worse and would evidently die if nothing were done. Free incision was made, four pints of seropurulent fluid removed, and drainage tube inserted. The next day the lung had expanded and in a few days the pleural cavity almost obliterated. In seven months the tube was no longer necessary.

West comments on the fallacy of the belief that in such cases the lung was bound down and could not expand. He does not believe that frequent. The idea that the pneumothorax was beneficial in curing the disease of the lung was refuted; the belief that operation in such cases hastened death was contradicted.

West advised incision (1) in the early stage if the dyspnœa and symptoms were urgent and not relieved permanently by paracentesis; (2) in the later stages if the effusion were permanent or some cases of obstinate serous pleurisy.

In the discussion Hall mentioned the rare cases in which the lung was bound down or was riddled with tuberculous cavities, in which cases the operation was not indicated. Dr. Thorowgood, Mr. Marmaduke Sheild, Dr. R. L. Bowles also spoke.

298. Leroux. Étude sur un cas de pneumothorax chez un tuberculeux. Epanchement pleural consécutif nécessitant la ponction. Guérison de l'hydropneumothorax. *La France Medicale*, XLIV, p. 561, 1897.

The patient was a girl, 19 yrs. old, with hydropneumothorax. The fluid was twice aspirated. The pneumothorax disappeared.

299. Bernard. Faut-il admettre un pneumothorax favorable. *La médecine moderne*, VIII, p. 449, 1897.

The writer begins by quoting Potain, who considered pneumothorax a complication of little moment, even a favorable feature, and Robin, who held the opposite view.

He studies the question on the pathological side. Czernicki's case, Forlanini, et al., are cited, showing the favorable influence of pneumothorax on the tuberculosis, hence he concludes that pathological anatomy shows this favorable result possible and ascribes this influence to

the compression of the lung. Clinically the evidence is against such good influence, although the case may for a while improve, but the pneumothorax case, with the respiratory function limited to one lung, is at the mercy of the least accident to this lung. The artificial pneumothorax as a therapeutic measure is now discussed. Potain is the first to voluntarily produce it (1888). Then in 1894 Forlanini produced it in two cases, using N for injection with good results.

In conclusion the writer thinks usually pneumothorax a grave accident, but in carefully selected cases (with unilateral lesions) it may be favorable and its production beneficial.

300. Bouchet. Pneumothorax tuberculeux avec pleurésie chronique. *Bull. Soc. Anat. de Paris*, LXXII, p. 453, 1897.

The case with autopsy is briefly reported. The point of interest seems to be that the perforation was through a very thick pleura. The fistula was at the posterior of the superior lobe, very small and resembled a hole made artificially with a needle.

301. Finney. A Case of Recurrent Pneumothorax. *Dublin Jour. of Med. Sciences*, April, 1898.

The case was that of a young man, 18 yrs. old, with no history indicating tuberculosis or any previous illness and no strain or violence, who on rising one morning felt a heavy weight at the top of the left chest. He tried to work, but must give up. On the next day he had neither pain nor dyspnœa; on the third day examination showed total left-sided pneumothorax, with hyperresonance, absence of respiratory murmur, slight metallic tinkling, fine bruit d'airain, no succussion. In forty-one days he was discharged well.

Two weeks later while at heavy work he felt a crackle in the chest, but noticed nothing more till the next morning, when sharp pain and dyspnœa called his attention to the return of the trouble. Again there was no liquid exudate. In twenty-nine days he was discharged well.

Finney calls attention to the amphoric respiration which on the first attack was present but disappeared on the fifth day (denoting the closure of the fistula?); to the absence of fluid; the metallic tinkling despite the absence of fluid; to the friction rub over the pericardial area of pleural origin and due to a reduplication of the pleura over the displaced heart; and to the short duration of the severe symptoms. He considers emphysema the cause, and reviews the few other such cases on record.

302. May and Gebhard. Pneumothorax durch gasbildende Bakterien. *Deuts. Arch. f. klin. Med.*, Vol. 61, 1898.

The patient, Andreas, 43 yrs. old, was stabbed twice, once above and once below the left nipple. The wounds healed quickly, but the temperature continued. A pleural effusion developed on the left side which was aspirated twelve days after the wounds, and a dark bloody fluid obtained which showed many diplococci. Gas now accumulated over the

fluid, and nine days later the pneumothorax was well developed. Succussion was obtained. Two days later (twenty-three days after the injury) the chest was again tapped and a bloody purulent fluid removed containing many gas bubbles, but without fetid odor.

The next day the pressure of the gas in the pleura was found to be + 12 mm. Hg. The gas burned with a blue flame. Three days later a rib was resected and the pus drained, but the relief was but temporary. Three weeks later two liters of fluid were removed from the pericardium. In ten days he died.

At autopsy were found subacute purulent fibrinous pleuritis and pericarditis following the stab wound, pneumothorax but probably no pneumopericardium.

The gas consisted of CO_2, H, and N. The isolated organism produced the same gas on media.

The sequence was supposed to have been, first, a hæmothorax. Then pus organisms produced an empyema on which the gas producing organism could grow.

The organism was not recognized but was thought not to be Frænkel's.

303. **Lieutaud.** (A Contribution to the Study of Pneumothorax and in Particular Concerning its Recovery.) Paris, 1898.

Conclusions: Pneumothorax, whether of tuberculous or other origin, can recover spontaneously if the exudate is purely gaseous.

If there is also a fluid exudate, spontaneous recovery is still possible, but takes a longer time, since the pleura absorbs gas with greater difficulty when its walls are covered with exudate.

When pneumothorax is well borne, intervention is useless and may be dangerous.

Thoracentesis should not be systematically excluded in the treatment of pneumothorax, but one should resort to it only when the dyspnœa amounts to asphyxia. In this case it may retard death or in cases facilitate recovery.

If hydrothorax supervenes, one should resort to puncture only in case of asphyxia and then we recommend to remove only a certain amount of fluid lest the pulmonary wound be reopened.

In case of a long-standing or stationary hydrothorax, successive paracentesis, removing small amounts each time. Thus one avoids the symptoms produced by a too sudden release of pressure from the lung.

The cure may be due to closure of the fistula by false membranes or cicatrization of the tear, or more rarely to a simple adhesive pleurisy like that following an operation for empyema.

The writer dedicates his thesis to Professor Dieulafoy. He reports one case of tuberculous pneumothorax with recovery which had been tapped twice, and collects twenty-seven cases from literature which give the data from which these conclusions were drawn.

The general subject is discussed at length.

304. Léger. (Contribution to the Study of the Treatment of Tuberculous Pneumothorax.) *Thesis*, Paris, 1898.

The author's conclusions are:

1. The treatment of simple tuberculous pneumothorax should be only symptomatic.

2. The great danger of pneumothorax lies in the infection of the pleura which occurs at the same time as the pneumothorax and which results from the penetration into the pleural cavity of infectious agents from the pulmonary lesion.

3. The treatment depends on the severity of the symptoms.

(a) In a moderate degree of infection it is only necessary to tap for an abundant or persisting effusion.

(b) In an intense infection one may (1) inject or (2) lavage with antiseptic fluids, best with iodine solution, or chloral or corrosive sublimate. If these injections or lavages fail, do a paracentesis as soon as possible, unless the pulmonary lesions are too far advanced.

4. To sum it up, the indications are the same as those for all infectious pleurisies. The pleurisy is the main thing, the pneumothorax only reduces the chance of complete recovery.

The writer of this thesis was a pupil of Potain, whose views he advances. The literature is good, the subject is well discussed.

In chapter 1 he considers the pure pneumothorax and discusses the value of paracentesis, the objections, the indications, and the method. Chapter 2 is a consideration of hydropneumothorax and paracentesis (especially the method of Potain), injections and lavages are discussed. Pyopneumothorax is the subject of chapter 3.

The value of these procedures is discussed in connection with eight cases which are reported.

305. Hnátek. (Experimental Investigation of the Circulatory and Respiratory Disturbances in Pneumothorax.) *Allg. Wiener Zeitung*, XLIII, p. 267, 1898.

The work of Guttmann, Leichtenstern, Weil and Thoma, Weil, Gilbert and Roger, Rodet and Pourrat, Lieven, Sackur and Aron is reviewed.

Page 279. His technic is described and the experiments on dogs with sharp cannulæ. His result is that the production of a bilateral pneumothorax raises the blood pressure and slows the pulse.

Page 291. The experiments with dull cannulæ, which causes less irritation.

Page 313. In conclusion he found, agreeing with Aron, that pneumothorax caused a slight rise in blood pressure in fourteen cases. The criticism made of former workers who used narcotized animals he avoids. The cause of this rise varies: The skin incision, the introduction of the cannula both reflexly raise the pressure, due to stimulation of the sensory nerves. But this rise in pressure is temporary.

The causes of the permanent rise in blood pressure he finds in the lack of O, and the dyspnœa. But these are not enough; another factor

causes the rise, and that is the entrance of air in the pleura. This was shown on curarized animals in which the influence of both the respiratory movements and the lack of O were excluded. But cut the vagi in these dogs, and the rise in pressure and slowing of the pulse due to the production of pneumothorax do not occur, since the direct stimulation of the air on the vagus does not reach the vasomotor and cardio-inhibitory centers.

306. Anders and McFarland. Pneumothorax with Discovered Perforation. *Proceed. of the Path. Soc. of Phila.*, Jan. 15, 1898.

The case was one of tuberculous pneumothorax with death soon after the onset, and the report of the early autopsy. There follows a review of the subject in which mention is made of the two cases of Herman Biggs in which pneumothorax followed the exploratory puncture of the chest with a hypodermic needle.

In the discussion:

Packard reported that he had seen a case in which an exploratory puncture of the chest was followed by sudden pneumothorax, profound shock and early death. At autopsy it was seen that the needle had perforated an emphysematous bleb of the lung.

Stengel said he had recently seen what he believed to be an aseptic pneumothorax produced in some way by paracentesis.

307. Osler. Spontaneous Pneumothorax. *Maryland Med. Journal*, 1898, Vol. 38, p. 461.

This case will be included among those reported in this paper from Dr. Osler's clinic.

308. Crowdy. Pneumothorax; Repeated Paracentesis; Incision; Recovery. *Brit. Med. Jour.*, Feb. 5, 1898.

This is the case of a tuberculous man, 34 years old, who "felt something give way in the right side," on turning in bed. Two days later the dyspnœa was so great that a fine cannula was introduced, and this was followed by general emphysema. The next day a permanent cannula was introduced. Later free incision was made. Complete recovery followed.

309. Hopkins. The Operative Treatment of Pneumothorax and Pyothorax. *Annals of Surgery*, XXVII, p. 216, 1898.

A case of pneumothorax, following a fractured rib, and one after a stab wound two inches long, through which the collapsed lung was visible, are briefly quoted. In each the chest was aspirated to remove as much of the air as possible by pumping, and the wound closed. In each the healing was very rapid (case 2, in twenty days). In empyema a valved cannula was used.

310. Hamilton. Pneumothorax, its Etiology, Symptoms and Signs, With a Study of Twelve Cases. *Montreal Med. Journal*, Dec., 1898.

The writer gives notes of twelve cases, and tabulates their important points. Six were probably due to tuberculosis (five surely), three to empyema, one to the Bacillus aerogenes capsulatus, and in two cases the etiology was uncertain.

The conclusions of the writer were:

1. There is such a form of pneumothorax as the non-perforative form.
2. Latent pneumothorax is not infrequent.
3. Occurring in the course of pulmonary tuberculosis, pneumothorax may have a retarding effect upon the disease.
4. Recovery of a total pneumothorax may take place without any sign of fluid.
5. In the advanced cases of pulmonary tuberculosis fluid is present, and the prognosis is grave.

The writer reviews briefly the etiology, especially of the non-perforative forms, quoting Jaccoud, Fagge and Powell, and especially E. Lévy, and May and Gebhard, whose, with his case IX, are three examples of pneumothorax caused by gas-producing bacteria. Hamilton also reviews Biach's statistics.

In the section on diagnosis the writer quotes Angereau on the latent cases, and discusses the value of Hippocratic succussion, percussion, and metallic sounds.

311. West. Intrapleural Tension. *Report of the Royal Med. and Chirurg. Soc., Brit. Med. Jour.*, May 14, 1898.

Dr. West emphasized the elastic contraction of the lung, the intrabronchial oscillation of from — 0.5 mm. Hg. on inspiration to + 1.5 mm. on expiration, and other physical factors governing intrapleural tension, as illustrated by twenty paracenteses on eleven cases of pneumothorax. In two the inspiratory pressure was 0, but in all the rest it was positive. A lengthy discussion followed. No data are given, only a brief society report.

312. Alexander. (A Valvular Trocar for Pneumothorax.) *Deutsche med. Wochensch.*, Nov. 3, 1898.

A proposed apparatus to insert in the chest of pneumothorax acutissimus cases which when the pressure is positive will allow the air to escape but not to enter, is proposed.

313. Hoover. Some Observations on the Physical Signs of Pneumothorax and its Treatment. *Cleveland Jour. of Med.*, Feb., 1898.

After a brief historical review the writer discusses the question of essential pneumothorax, and says "with the single exception of Lévy's case, I can find no instance in which the proof of an essential pneumothorax rests on a complete clinical observation."

The elasticity of the lungs is then considered, and its difference during life and after death.

The effect of the varying intrapleural tension on the clinical symptoms and physical signs is well shown by his cases and experiments on the cadaver and on animals. The vocal fremitus over the air-sac was absent in but one of his seven cases. The percussion note depends on the size of the air cavity, the thickness of the thoracic wall and the tension of the gas contained. The greater the pressure the higher is its pitch and the shorter its duration, and it may resemble that over fluid rather than air. It may also acquire a metallic character. By his experiments on this point it would seem that the above was not well confirmed, for he says: "The living human subject with a pressure-pneumothorax seems to increase the rigidity of the thoracic wall by muscular action and in this manner modify the percussion note and increase the resistance in a manner that cannot be reproduced experimentally on lower animals when alive, nor in the dead human subject."

He thinks the coin sound depends not on the tension of the gas but on the tension of the walls of the cavity and gives experimental evidence of this view.

That the high pitched amphoric character of the breathing is due to the consonance of the air-sac and not to an open fistula he considers proven, and adds a very good case in point: a typhoid patient had a considerable distension of a loop of intestine that lay in the left hypochondrium. When the patient lay on his back there could be heard an amphoric consonance accompanying the normal respiratory sounds. When he turned on the left side this disappeared and only the vesicular sounds were audible.

The absence of respiratory sounds in a case of pneumothorax is hard to explain. There must be either a lack of tension in the walls bounding the pneumothorax cavity, or a thickening of the pleura, or lack of consistency of the compressed lung which impairs the conduction of the sound.

314. Levison. Beitrag zur Casuistik des Pneumothorax bei Gesunden. *Münch. med. Wochenschr.*, Oct. 10, 1899.

Levison reports the case of E. N., a man, 19½ years old, with goitre, who one noon climbed a mountain and suffered from shortness of breath. Eight hours later, after a hearty laugh, he felt something in his right chest tear, followed by dyspnœa. In ten days the chest was normal.

The author discusses similar cases reported. Dowel's case was the only other one he found following laughing. He explains his case as caused by the rupture of an emphysematous bleb.

315. Rose. Über Verlauf und Prognose des tuberkulösen Pneumothorax. *Deut. med. Wochensch.*, Oct. 26, 1899.

In that hospital (Bethany, Berlin) they have had 22 cases of pneumothorax, of which 19 (86 per cent) were tuberculous in origin. Of these 19 cases 3 were women. 15 died of the pneumothorax. He analyzes the cases further and reports several of them.

Continued, Nov. 2, 1899.

The whole is a good review of the subject.

316. Drasche. Aetiologie des tuberkulösen Pneumothorax. *Wiener klin. Wochenschrift*, Dec. 21, 1899.

For more than forty years of hospital connection the writer has been interested in tuberculous pneumothorax, and reports 198 cases, of whom 158 were men and 40 women—the largest number yet reported. These composed 2.36 per cent of the men, and 1.13 per cent of the women treated there for tuberculosis. Weil's computation that 8.10 per cent of tuberculous cases develop this condition, must be based on too few cases. The maximum number of cases were during the third decade, when lung tuberculosis is most common and dangerous. 55 per cent of the cases were on the right side and 44 per cent on the left. This is opposed to all previous statements. There were 3 cases of double pneumothorax, one died at once, one lived two hours, and one twelve hours after the bilateral involvement. Among 318 autopsies on pneumothorax cases made at this hospital, there were 11 cases of the double form. These might be agonal or post-mortem cases.

Ten were cases of partial pneumothorax, the lung being quite adherent, and the pneumothorax cavity having the form of a cone, the base being the costal pleura, the apex, the perforation. The lung was compressed only in the immediate vicinity of the cavity. Four were cases of *pneumothorax bipartitus*, the cavity being divided into two parts by a false membrane which communicated by slits in this partition. The physical signs of these cases are particularly interesting.

Cause.—Of the 198 cases, 34 followed coughing. Other cases dated it to straining at stool, vomiting, lifting heavy loads, and one to turning over in bed. He remarks on their occupations.

In 71.5 per cent the perforation was in the upper lobe, but beyond this there was no especial predilection of location. In but 17 cases were there multiple perforations.

Only exceptionally (in rapidly fatal cases) is a liquid exudate absent.

Other interesting points are mentioned in this brief but interesting and important paper, it being the largest list recorded by one observer.

317. Stembo. Beitrage zur diagnostischen Verwerthung der Röntgen Untersuchungen. *Deut. med. Wochenschr.*, 1899, p. 28.

Hydropneumothorax. Two cases are reported and the skiagrams given, showing that the latter alone is enough for diagnosis.

318. Delafield. Pneumothorax. *The Tri-State Medical Jour. and Pract.*, St. Louis, Vol. VI, p. 14, 1899.

The man had had some history of cough, but was in apparently good health. One day he was suddenly seized with dyspnœa and felt "as if his spine was squeezed in a vise." This attack came on while he was

quiet at the table. The symptoms diminished until in four days there was dyspnœa only on exertion. Three days later a dry cough began, and two days after this he entered the hospital. The right chest was distended; the heart displaced to the left. The change of the percussion note was slight, that of the right side being "not so distinctly pulmonary" as on the right. On the right side everywhere amphoric breathing was heard. No fever, no pleurisy followed. The writer considered the cause tuberculosis.

319. Finley (F. G.). Pneumothorax From Gas-Producing Bacteria. *Montreal Med. Jour.*, Vol. 28, p. 759, 1899.

In a brief historical note on pneumothorax without perforation of the pleura, he ascribes to Senator (*Deut. Zeit. für Med.*, II) the opinion that gases held in solution may be liberated if the pressure under which they are be diminished by retraction of the lung, but such an amount must be inconsiderable; further he held that purulent fluids undergoing decomposition in the pleura can evolve gases, and supports this argument by the subcutaneous emphysema in the absence of an external wound. Welch's case of pneumoperitonitis (*Jour. Exp. Med.*, I) due to the Bacillus aerogenes capsulatus, placed such observations on a scientific basis.

The case is reported of a man, 46 years old. At first he had severe pain in the abdomen. Some days later clear serous fluid was found at the right base, and later (how long?) right-sided hydropneumothorax was discovered with all the clinical signs of that condition. Four days later he was aspirated and 20 ounces of almost pure blood obtained, which clotted at once. The patient died seven days later. The clinical diagnosis was localized pneumothorax at the base of the lung. No tubercle bacilli were found in the sputum. At autopsy a sub-diaphragmatic abscess was found communicating with a localized pyopneumothorax cavity at the right base of the pleura. The anatomical findings excluded the possibility of a perforation into any of the hollow organs, hence he thinks certainly the gas was due to the Bacillus coli found in the exudate.

He quotes at length and reviews the three other similar cases on record, Levy's, Nicholls' which he considers the clearest case, and May and Gebhard's.

He concludes that pneumothorax due to gas-forming organisms, Bacillus coli and Bacillus aerogenes capsulatus does rarely occur. And if a gas foreign to the atmosphere (e. g., hydrogen) is found it is evidence of such a case.

320. Finlay (D. W.). Pneumothorax. *Allbutt's System of Medicine*, Vol. VI, p. 378, 1899.

On the autopsy table he had seen it but twice in 60 cases of phthisis. He calls attention to the fact that the downward displacement of the liver forms a large pocket for fluid, hence the actual amount may be underestimated. The cracked pot sound has been reported present when the

fistula is open. The amphoric respiration is due to the air passing through a patent fistula and is most likely to be easily detected just over the site of the perforation. In a foot-note concerning metallic tinkling **Allbutt** says that in one of his cases the tinklings could be heard by the medical attendant for about two hours in all parts of a large room. They were much more frequent at first and were more numerous and louder on inspiration. The rupture of the lung was brought about by an attempt to bend the body backwards so as, if possible, to touch the ground with the hands without removing the toes from a line. The patient soon got well and remained well. The patient is described as a healthy and athletic young man. Finlay suggests they were due to a rupture of a tiny bubble at each issue of air.

Under treatment he advises paracentesis if there be considerable positive pressure in the chest, and strapping of the affected side to prevent subsequent distension.

If the fluid be purulent he recommends operation on general principles, but states that tuberculous pyopneumothorax cases seldom recover.

He agrees with Thompson concerning the prophylactic measures to be attempted in a tuberculous case with threatened perforation of the lung.

321. Williams. Observations on Pneumohydrothorax and Pneumothorax. *Med. and Surg. Reports of the Boston City Hospital*, p. 191, 1899.

These four cases were studied with the Röntgen light, which permitted the determination of the exact conditions and positions of the intrathoracic organs. Even the waves made on the surface of the fluid by the heart-beat were visible. In one case aspiration of air was followed by immediate and permanent improvement. Diagrams of the chests of the patients are given.

321a. Villy. A Case of Pneumothorax in a Young Child; no Primary Lesion Being Discovered. *The Medical Chronicle*, 3rd S., I, 1899.

Pneumothorax in young children is, he believes, generally due to the rupture of an abscess of the lung into the pleural cavity, and mentions two such cases which he had observed.

The present case was without discoverable lesion, and, he is confident, was due to an overstrain of the lung and pleura in an attack of coughing. The child was 2 years old, a convalescent from diphtheria and subject to frequent paroxysmal attacks of coughing. Twenty days after admission there suddenly developed extreme dyspnœa, much cyanosis, and "recession of the lower thoracic parietes." "The entry of air into the lungs appeared to be only slight at any point, whilst at the angle of the scapula the breath sounds appeared to be distinctly weaker than at any other place, and as on admission there was impaired percussion resonance," a needle was inserted three times, but the syringe "produced nothing but an escape of gas while the point was plainly felt to have entered a large cavity. Next day amphoric breathing was present over the upper and anterior part of the right lung; subcutaneous emphy-

sema had developed round the punctures already made. The right side of the chest became hyperresonant; the liver was pushed down and the cardiac dulness displaced entirely to the right of the sternum. Later amphoric breathing extended to the back of the chest; the bell sound and metallic tinkling were also found." Two days later a portion of rib was resected, no pus found, but air rushed out. She died six days after the onset. At autopsy a complete, pure pneumothorax was found. After removal, a point where air escaped was demonstrated in the fissure between the lower and middle lobes. Except for a pleurisy and collapse the lung was not diseased.

We report this case at length, since had it happened at our hands we would have reported it as a case due to puncture.

322. **Phalip.** Du pneumothorax accidentel survenant au cours de la thoracentèse; et du Pneumothorax artificiel créé par injections d'air ou d'oxygène dans la plèvre. Paris, 1900.

This thesis was written from Bard's initiation.

Conclusions:

1. Pneumothorax, more or less grave, can result from thoracentesis, but it is not just to blame this alone, since the accident would often not occur if there were not a pathological condition of the lung and pleura.

2. The only cause he recognizes is a tear of the lung and pleura. The wound of the lung by the needle can rarely do this, and a spontaneous origin in the pleura is true only if organisms produce the gas.

3. To avoid this accident the pleura should be aspirated carefully, and not all at one time.

4. Especial precaution should be used in hydrothorax following pneumohydrothorax to prevent recurrence.

5. In some cases of tuberculous effusion it might be well to substitute carefully sterilized gas or air for the fluid.

The writer mentions four possible causes of the penumothorax.

1. Wound of the lung by the trochar.
2. Entrance of external air.
3. Liberation of gas in the effusion.
4. Ulceration or tear of the lung.

Illustrations of each are given, or those given by others criticised.

The production of artificial pneumothorax as a therapeutic measure receives a long consideration, and many illustrations are given of the result. The technic is well described.

323. **Desbiez.** (Valvular Pneumothorax.) Paris, 1900.

This thesis, inspired by Béclère, begins with a review of the subject, and contains a description and figure of Béclère's simple manometer with which to test the intrapleural tension. Three cases are reported in which this instrument was used, but he gives no definite figures of the pressures found.

His conclusions are: (1) Valvular pneumothorax is a variety characterized by an intrapleural tension above that of the air. (2) It is a clinical form deserving of attention. (3), (4) For the production of this positive pressure the efforts of cough are necessary. (5) This form of pneumothorax is to be suspected when the dyspnœa increases, also the cyanosis and symptoms of asphyxia, the exaggeration and extension of the tympany, the displacement of the heart, and if on the right side, that of the liver, become greater. (6) But the only sure sign of valvular pneumothorax is the determination of a positive intrapleural pressure, and this is a sign of bad prognostic import. (7) Béclère's manometer is the easiest instrument with which to measure this pressure, and (8) this instrument allows also the superfluous air to be removed, (9) better and more safely than by aspiration. (10) Morphia is especially indicated in these cases to diminish the cough. (11) When these capillary punctures do not suffice a permanent cannula with a sterile cotton plug to filter the air is indicated. (12) Radioscopy is recommended as an excellent method of following these cases.

324. Beclère. (The Diagnosis and Treatment of Valvular Pneumothorax.) *Bull. et mém. de la Soc. méd. des Hôp. de Paris*, 3 series, Vol. 17, p. 445, 1900.

A true valve seldom exists. It is the coaptation of the sides of the orifice or the collapse of the walls of a long narrow fistula which furnishes the mechanism. That the pressure in these cases may rise above that of the atmosphere, it is necessary that the patient cough, and with effort. In valvular pneumothorax the lung is not only collapsed but compressed, and the pressure symptoms on the heart and the other lung are comparable to those produced by a liquid effusion; they are increased with each spasm of coughing. There lies the so great danger of valvular pneumothorax. These symptoms he reviews.

To measure this intrathoracic pressure he proposes his instrument above described.

The value of the instrument he has been able to prove in cases of open and valvular pneumothorax, both for diagnosis and for treatment. He thinks it sufficient for the treatment; that aspiration should not be used, since it may reopen the fistula if closed, and is useless in open and valvular. Capillary punctures are simple, can be repeated, and avoid the danger of general emphysema following the use of the trochar.

This instrument, with morphin to quiet the cough, he thinks sufficient in treatment.

Gaillard considered (in the discussion following the paper) the instrument useful in cases of pure pneumothorax, but these cases of suffocative pneumothorax are rare, hence there may not be much occasion to use the instrument. In valvular pneumothorax, on the other hand, the danger is not so much from the air of the affected side as from the pulmonary lesions of the opposite side. The patients in whom the unaffected lung is sound generally survive even without puncture.

325. Duplant. (Valvular Pneumothorax.) *Revue de Méd.*, 1900, p. 730.

The writer studied two such cases carefully. He proposes to speak only of the importance of adhesions in the etiology, and of the valvular mechanism.

Years ago, Tripier noted the constant presence of adhesions near the orifice of the fistula; Duplant agrees; he thinks that at onset a tuberculous pneumothorax is almost always partial.

He agrees with Desbiez and Bouveret as to the importance of cough in overcoming the resistance of the valve. But a true valve is rare and unnecessary, for the compression which the collapsing lung exerts on the fistula is sufficient.

He reports two cases illustrating the above points. The pressure in the pleuræ was positive.

Thanks to the Röntgen rays and to inspection he could determine that in pneumothorax accompanied by complete collapse of the lung, the thorax is absolutely immobile and the diaphragm completely inert. Only if this were true could the cough in valvular pneumothorax fill the chest; otherwise the cough would raise the pressure in each side equally and the valve could not open. The valve action is present only when the lung has collapsed, the valve does not aid in forming the pneumothorax but increasing the tension of a pneumothorax already formed. At first it is during inspiration that air enters.

Conclusions (omitting the points mentioned above):

1. Adhesions of the pleura do not oppose the production of pneumothorax. The tear comes near an old organized adhesion which rendered the lung at this point immobile, hence the strain of coughing tears it; also it is weakened here by the tuberculous process.

2. The frequency of adhesions in tuberculosis explains the frequency of partial pneumothorax of tuberculous origin at the first, which becomes total as the air disengages these adhesions. This can be followed clinically.

3. The great majority of tuberculous pneumothorax cases are valvular, but the valve exercises its function only after the lung has completely collapsed and the thoracic walls on that side are immobile.

326. Reineboth. Die Valsalva'sche Versuch bei geöffneter Brusthöhle ein mass fur die Wiederausdehnungsfähigkeit der Lunge beim Pneumothorax. *Deuts. Archiv f. klin. Med.*, 1900, p. 308.

The "Valsalva effort" is made by taking a deep breath, closing the glottis, and then making a forced expiratory effort. The effect is to compress the air in the lungs, venous stasis, and expulsion of the blood in the lungs to the heart. This is indicated in the pulse tracings. But if the chest be opened, part of the effort is expended in distending the collapsed lung, if it can be expanded; the venous obstruction is less, and the base line of the pulse curve rises less or none. But if the collapsed lung cannot be inflated, the pulse is affected as under normal conditions.

The writer gives several cases, the clinical histories and the pulse tracings of which seem to bear out his conclusion that this is a valuable method, especially after the radical operation for empyema, to test the expansibility of the lung. We must know, however, the size of the cavity. If, however, there be a fistula between the pleura and bronchial tree, the method is unreliable.

327. Aron. Die Mechanik des geschlossenen Pneumothorax beim Menschen. *Virchow's Archiv*, No. 161, p. 569, 1900.

This case is also reported in his pamphlet of 1902, to the abstract of which (358) the reader is referred.

328. Hoppe-Seyler. The Composition of the Gases Present in Pneumothorax. *Deuts. Arch. f. kl. Med.*, 1900, p. 105.

He used Hempel's gas absorbing pipettes in examination of twelve specimens of gas taken at various times from four patients.

In Case 1 three analyses were made, and O was never found (the third analysis was $CO_2 = 11.4$ per cent, $N = 88.6$ per cent).

In Case 2 on one day no O was found, on other days considerable (e. g., Anal. 1: $CO_2 = 5.6$ per cent, $O = 4.5$ per cent, $N = 89.9$ per cent). He explains the absence of O by the long time the gas remained in the chest and the presence of an exudate with organisms whose growth would consume the O.

Case 3 is a closed pneumothorax with a decomposing exudate. The $CO_2 = 20.4$ per cent (probably from the decomposition), $O = 0.3$ per cent, $N = 79.3$ per cent.

Case 4, also with decomposing exudate, shows the maximum CO_2 on record (49 per cent), $H = 21.6$ per cent, $N = 29.4$ per cent. This is first reported determination of H in the gas.

Among the factors governing the composition of the gas are:

1. The selective action of the pleura, hence the readiness with which O is absorbed; therefore in the phthisis cases the O per cent is low, or 0; the N per cent is high.

2. The presence of bacteria.

The tissue also "excretes" CO_2, 5 to 12 per cent, and although such an excretion of N it is not necessary to assume, yet it does not occur when the gas introduced contains little or no N. The bacteria use up O and produce CO_2, and by the metabolic processes in the decomposing masses of pus CO_2 is produced and O consumed, and even reduction processes may occur and H be produced.

He presents his analysis in tabular form.

329. Boland. Idiopathic Pneumohæmothorax, with Recovery after Aspiration. *Boston Med. and Surg. Jour.*, Vol. 142, p. 321, 1900.

The case is reported of a man 34 yrs. of age. His health had always been good. Of recent years he has worked as a marble cutter, but gave this up, since he had some symptoms of lung trouble.

The family history is negative as regards tuberculosis. One evening while in his usual health he woke up feeling a very severe pain in the right chest and a sensation as if wind were escaping there. The next morning all the signs of right-sided pneumothorax were present. The patient improved somewhat, but at the end of 21 days it was decided, since effusion was accumulating, to aspirate the chest, and 34 ounces of dark blood escaped. No air was withdrawn. Fifteen days later, as the fluid had increased, a second aspiration was performed and 39 ounces of dark blood escaped. This blood was almost pure. The author considers the case an exceedingly rare one, and calls attention to the fact that the uncoagulated condition of the blood confirms Pagenstecher's opinion that the pleural cavity has the power to prevent coagulation. He considers this case idiopathic hæmopneumothorax, due possibly to an old pleurisy, the adhesions of which were torn by the movements of stretching which the patient made before retiring that night.

330. Rumpel. *Deuts. med. Wchs.*, Vol. XXVI, pp. 248 and 256, 1900.

The writer reports his method used in making frozen cross sections of the body for the study of the topographical anatomy of the chest in pneumothorax and empyema.

Discussion.—In this the value of Perthes' apparatus is discussed.

331. Villy. A Case of Pneumothorax Complicating Whooping-cough. *Medical Chronicle*, Vol. III, p. 99, 1900.

The child was 3 yrs. old, admitted June 16, 1899, for a severe attack of faucial diphtheria complicating whooping-cough, and was recovering from an attack of measles. The faucial condition cleared up rapidly, but the attacks of coughing became very frequent and severe, but until July 7 the lung signs were negative; the râles were heard over both lungs, especially at the bases, and several small areas of rather doubtful impaired resonance (broncho-pneumonia) were noted. July 15 the child was exceedingly ill, pulse 145, respirations 80, with very severe spells of coughing, and the physical signs, although not much changed, were more marked at the left base. July 17, slight hyperresonance seemed present at the left base. A needle was inserted at the angle of the left scapula and only air withdrawn. This was also true July 19, when aspiration was attempted and no pus was obtained. The child improved and was discharged Nov. 4.

In this case the description of the physical signs is very indefinite. The child had diphtheria with the whooping-cough and it is doubtful which explained the pneumothorax.

332. Weber. Pneumothorax without Effusion in an Apparently Healthy Man. *Trans. Clin. Soc. London*, Vol. 33, p. 171, 1900.

The patient, 31 yrs. old, gives a good family and past history. Nov. 21, 1899, while doing ordinary work (pianoforte fitter), he was seized with a knife-like pain to the left of the sternum. He was dyspnœic, but

able to walk to the hospital. About one-half hour later, respirations 30 to 32; respiratory movements equal; cardiac dulness to the right of the sternum; percussion note tympanitic over the lower left thorax; no respiratory sounds on the left side; the coin sound was present.

Nov. 23.—Physical signs typical; no succussion splash.

Nov. 25.—The movement and vocal vibration on the left side are diminished. Over the whole left side the breath, voice and cough are of amphoric quality. There is distinct bell sound, but no metallic tinkle or splash; resp. 20.

Nov. 30.—The bell sound cannot be heard over the upper and inner portion of the left chest and to the left of the sternum.

Dec. 14.—The bell sound is now heard over the lower left front.

Dec. 21.—No bell sound anywhere.

The duration of the case (from onset to the return of the heart to the normal position) was thirty-two days. The subjective symptoms were slight. There was never a sign of fluid in the chest. The gradual disappearance of the bell sound was evidence of the expansion of the lung. After this has disappeared the voice sounds and fremitus were still diminished. The return of the heart was the last sign to indicate recovery.

Similar cases are now discussed. The writer states that his father told him of pneumothorax in an apparently healthy man recurring after two years. He recovered from both in a few weeks. As to the cause of such cases, the fact pointed out by Whipham and Hall that the age of most of the patients with this form is under thirty years, is evidence of its tuberculous nature. He discusses the emphysematous cases.

333. Pitt. A Case of Rapidly Fatal Hæmopneumothorax, apparently due to the Rupture of an Emphysematous Bulla. *Trans. of the Clin. Soc. of London*, Vol. 33, p. 95, 1900.

The patient was a young man, 18 yrs. old. His past history was good. The present illness began Sept. 15, 1897, with sore throat and vomiting, but after one day in bed he went to work. He was quite well Sept. 19, and took a walk, but soon returned because of pain in the right shoulder and chest, which gradually increased, until the next morning he was found mortally ill, cold and pulseless. He was profoundly anæmic. On examination he obviously had a pneumothorax with fluid. The right side was tympanitic in front, but dull below the anterior axillary line. In front were the bell sound, distant tubular breathing, and diminished voice sounds. The heart was so displaced that its maximum impulse was two inches to the left of the nipple. A needle was inserted and blood escaped. He died that night.

At autopsy was found "eight pints of fluid blood and enough clot to fill the hands three times, together with a considerable amount of air under great tension. . . . The lungs were absolutely healthy with the exception of a small projecting emphysematous bulla about half an inch across near the right apex, the wall of which was imperfect, and attached to this was a fibrous band of the thickness of a knitting needle, which

had been torn across. The blood may have come from a vessel in this adhesion, but no aneurysmal pouch or obviously patent vessel could be seen." There was no trace of tuberculosis. This is the first such case on record, with the exception perhaps of a case to which Saussier refers.

The causes of hæmothorax, the fate of the blood, and the causes of pneumothorax are then discussed. There are six causes for both. Of these, trauma, phthisis, gangrene, hydatid and cancer can be excluded. The pneumothorax in this case was surely due to emphysema, but the cause of the hemorrhage he could not state.

Appendix.—The fatal cases of pneumothorax and hæmothorax at one hospital between the years 1885-1898 are reported. Pneumothorax was found in 28 cases—phthisis, 17; laceration of the lung, 4; tracheotomy, 3; pulmonary thrombosis, 1; necrosed pulmonary tissue, 2; rupture of an emphysematous vesicle, 1. Three were cases of hæmopneumothorax—1 of rupture of a large tuberculous cavity and opening of a large vessel, 2 were cases of children with rupture of the lung from being run over, without fractured ribs. There were during the period 65 cases of hæmothorax. These are classified. Notes are given of each of the 28 cases of pneumothorax.

334. **Béclère.** Diagnostic et Traitement du Pneumothorax à Soupape. *La Presse Méd.*, 1900, p. 197.

The method of production, importance and danger of valvular pneumothorax are described. For its diagnosis and treatment he recommends his simple instrument.

335. **Variot et Roy.** Pneumothorax et vomique séreuse au cours d'une thoracentèse chez une petite fille de onze ans. *Bull. et mém. de la Soc. méd. des Hôp. de Paris*, 3 s., XVIII, 1040, 1901.

They consider such cases excessively rare.

The child had persistent effusion of the right chest and was punctured twice, the Pravaz syringe being used, unsuccessfully. Then the Potain apparatus was used. Air escaped, and clear serous fluid. Severe coughing ended the tapping and continued with increasing vehemence until the child expectorated an abundance (about 250 gms. in 15 minutes) of bloody serum, and asphyxiation seemed imminent. At once loud amphoric respiration was heard, amphoric cough, and hyperresonant percussion note. In twenty-three days the child was practically well. In discussing the case the writers consider it a case of partial pneumothorax. They deny it was of traumatic origin, since they saw no drop of blood escape by the needle. The cause was probably a tear of the weakened lung tissue, due perhaps to an adhesion, the result of the long-standing pleurisy. They think the sputum was the pleural exudate.

Galliard, in the discussion, says in such cases one need not blame a wound of the lung by the trochar, nor formation of gas from the exudate, but the rupture of the lung at a weak point. He thinks in the above case the sputum was simply the albuminous expectoration of paracentesis.

Rendu questioned the past history of this case, and asked, Could not the pneumothorax have preceded the aspiration? He had one case of a man with the signs of pleural effusion, aspiration, and then the signs of pneumothorax, but without symptoms. He then examined the past history of the case and found a quite clear history of preceding pneumothorax. This had improved and cleared up as the effusion developed, and the tear was reopened by the puncture.

Variot admitted his case might have been one of cured pneumothorax.

336. **Mayer.** PEUT-IL PRODUIRE APRÈS THORACENTÈSE UN PNEUMOTHORAX EX VACUO. *Revue méd. de la Suisse Romande*, Vol. XXI, p. 112, 1901

Since the use of aspiration to evacuate a pleural exudate, pneumothorax has been found to follow its employment, some at once, some a few days later. This is not surprising in the case of tuberculosis, but is when no fistula or scar of one can be found, i. e., in "spontaneous" cases.

The opinions of Itard, Laennec, Saussier, Stokes, Wunderlich, Jaccoud, Boisseau, West, Niemeyer, and others concerning these cases are quoted. West first advanced the idea of pneumothorax ex vacuo, the decompression caused by aspiration changing some fluid into the gaseous condition. Bard thought one case due to exhalation from the capillaries of the pleura from repeated aspirations. The writer, under Bard's direction, reviews the literature of the question. He excludes all cases in which it is probable the lung was wounded, or air entered from outside, or was due to bacteria inside the pleura, and found but three cases besides Bard's. Bard's explanation is first quoted; the long compressed lung cannot expand to fill the space of the evacuated fluid, hence the gas in the blood of the pleural capillaries is liberated. That such gases are given off by the blood is seen in the change of the gas mixture in a pneumothorax after injecting a pure gas.

The cases accepted are of Niemeyer, Béclère and Bucquoy.

Bard's Case.—That of Bard is reported. The patient was repeatedly aspirated, large amounts being removed. In this case it developed twice after puncture. The case receives considerable criticism from the writer.

337. **Roy.** PNEUMOTHORAX WITH VOMICA FOLLOWING THORACENTESIS. *La Semaine Méd.*, Oct. 10, 1901.

A girl, 11 yrs. old, was tapped for an old pleural exudate which would not resolve; 120 oz. of serous fluid were obtained, after which she expectorated a fluid like that aspirated, and pneumothorax also followed. The diagnosis made was pneumothorax with tuberculous cavity following tapping, due probably to a rent in the lung by an old adhesion. Perfect recovery.

Galliard said he thought pneumothorax often followed the rupture of pleural adhesions after tapping, and that the sputum in the above case was the "albuminous expectoration."

338. **Stiller.** (PNEUMOTHORAX FROM SNEEZING, THE LUNGS BEING SOUND.) *Wien. med. Wochenschr.*, May 4, 1901.

The patient was a healthy young man, who was seized with unbearable gastric pain following a fit of sneezing. The dyspnœa was extreme. The diaphragm was everted into the abdomen by a right-sided pneumothorax. There was no metallic tinkling or succussion. Perfect recovery in one week.

Stiller considers this the only case on record in which the pressure of the air alone had forced down the diaphragm, dislocating the liver; hence it was a case with a perfect valvular fistula.

The sudden abdominal pain he ascribes to rupture of the suspensory ligaments of the liver.

The writer discusses such cases, maintaining that this case had perfectly normal lungs.

339. Hayem. Pneumothorax. *La Tribune médicale*, 2 s., XXXIII, p. 545, 1901.

This is a clinical lecture, with the presentation of one case. He says absence of vocal fremitus and absence of breath sounds are the two most important signs. The tympanitic note is dulled by false membrane on the pleura and by increased tension of the gas.

340. Martin. A Case of Pneumothorax shown by the Roentgen Rays. *Lancet*, Sept. 28, 1901.

Martin thinks this the first skiagram of this disease reported (but later finds Béclère's case and others). "The photograph is of interest as showing more accurately than physical signs could demonstrate the actual condition of affairs within the thorax and the displacement of the viscera."

The following paper is so important that its review will be especially minute.

341. Bard (Prof. de Clin. Méd. à l'Univ. de Genève). Recherches experimentals et cliniques sur la Pression intrapleurale dans le Pneumothorax. *Revue de médecine*, Vol. 21, 1901.

I. For a long time it has been considered an important point to determine whether in pneumothorax the fistula had closed or not. Weil's opinion has been generally accepted. He said, determine the intrapleural pressure; if positive, aspirate, and if the pressure rises again, it is open; if it becomes positive at the end of expiration, it is a valvular pneumothorax. But it does not seem that he ever used this method. The author then cites the measurements of pressures made by Powell, Weil, Quincke, Seifert, Meunier, Pitres, Béclère, Duplant, adding, "Such is all the accurate information I could discover in literature," but rules out all cases in which fluid was also present. All since Weil have explained a positive pressure as the results of a valvular fistula and failing so often to find an anatomi-

cal valve have given various explanations of the mechanism replacing it. But "it is singular that no one has sought to explain why the length of the fistula, the sinuosity of its course, the pulmonary retraction would give a valvular effect without closing the lumen altogether . . . and why it is an obstacle (to the passage of air and fluid) always in the same direction." But even though most admit of the presence of valvular action, they do not agree at all as to the mechanism; normal inspiration is the force which raises the pressure according to most Germans, forced expiration (e. g., coughing) according to Bouveret and others, but all agreed that a positive mean pressure was evidence of some valvular arrangement of the fistula.

II. One day while using Béclère's simple apparatus in a clinical demonstration of a case of pneumothorax Bard found a mean positive pressure of 4 cm. of water with inspiration and 8 cm. with expiration. Coughing forced air out of manometer, after which the pressure oscillated around 0, and in a few minutes returned to the former level. He pinched the tube of the manometer during the coughing to prevent the expulsion of air; on opening it coughing had not raised the pressure; it was either the same or lower than before. Hence neither of the prevalent theories are satisfactory. The positive pressure is not due to inspiration, since after lowering the pressure by expelling air the positive pressure slowly re-established itself even after inspiration had ceased to create a negative pressure in the cavity; nor is it due to cough, since after the temporary disturbance due to the effort the pressure was found unchanged or lowered, and in the latter case slowly rose without the intervention of coughing. The slight lowering could be due to expulsion of air through the pulmonary wound, or greater collapse of the lung, thus increasing the size of the cavity. He thinks the latter.

III. Bard now states the theory which he proposes to prove: The regular play of quiet respiratory movements tends to raise the intrapleural pressure to a constant fixed level. This level, + 7 to + 8 cm. of H_2O, is so constant that it cannot be due to such a variable cause as coughing, etc., and is about that tension ascribed to the elasticity of a normal lung. He cites estimates of this elasticity. The pleural cavity and the bronchial tree in pneumothorax are two communicating cavities; the tension of the gas in each will strive to come to a state of equilibrium which is simple if the fistula be open in both inspiration and expiration; but if valvular then the pleural cavity will take the maximal of that of the bronchial tree. These two cavities are subject to the same forces, but these forces are equal in direction, not in magnitude. The tension in the pleural cavity is governed by the chest walls, while that in the bronchi by the chest walls plus the elastic tension of both lungs, the sound lung and of the collapsed if any be left; hence soon the intrapleural pressure will be equal to the former plus that of the elastic tension of the lungs such as this may be. In forced expiration the pressure in each will be raised, but in the pleura more than in the bronchial tree (because of the intervention of

the lung), hence air will be forced out through the pulmonary wound if not valvular, thus lowering the pressure, or the same would result from compressing the lung (thus increasing the size of the cavity). Air can enter the pleura both during inspiration and expiration, according to the difference of tension in both cavities. When the intrapleural tension is negative this can occur during inspiration, but after the tension is positive during inspiration it can enter only during expiration and then only until the positive tension in excess of that of the atmosphere is equal to the elasticity remaining of the lungs. During cough expiration will add no air, since the difference between the tensions of the two cavities is less then than during quiet respiration.

IV. Animal experiments are now described. "A long series of observations were made on rabbits and each repeated a great many times until I was perfectly sure of the constancy of the observed phenomena." He criticises the work of Weil, who neglected two important elements, (1) the rapid absorption of gas in closed pneumothorax, which lowers the tension, and (2) if the fistula be open, the variable factor of the bronchial-tree pressure, which is never just 0 as an average. (3) In valvular pneumothorax Weil says the tension will be positive with expiration and negative with inspiration, but since that of the bronchial tree into which the pleura opens is positive, that of pleura at the end of inspiration will also be. Weil's experiments were all cases of external pneumothorax, and the element of the lung wounds was not considered, hence his results have little clinical value where the wounds are through the lung.

A manometer is inserted into the chest wall of rabbits. Bard found — 3 to — 4 cm. H_2O pressure with expiration, — 8 to — 10 cm. with inspiration. If the side tube of the manometer be open the pressure oscillates feebly about 0. If air be forced in through this tube the pressure may be raised to + 2 to + 3 cm. H_2O with inspiration and + 5 to + 6 cm. with expiration, but beyond that the animal struggles. With the pressure negative the animal is quiet, but if the pressure is positive the convulsive movements of the rabbit force air out through the manometer until negative tension is restored. If it fails to force the air out the pressure soon begins to slowly fall, so that in one-half to three-quarters of an hour it will be negative (due to absorption of air). The harder the animal struggles the quicker the pressure falls, which may be partly due to compression of the lung, and none he thinks to leakage.

This preliminary series of experiments involved conditions in which there was no pulmonary wound, and showed that the efforts of the animal despite the collapsed condition of the lung force enough air from the pleura to restore negative tension.

These experiments were repeated with the presence of a pulmonary wound. The wounds were made in various ways, cutting, tearing, burning, etc., and of various extent, from a limited wound to the removal of half a lobe, *but the results were all the same, provided the wound was limited to the parenchyma of the lung*, and did not involve its pedicle. These animals (with pulmonary wounds of various character and extent)

acted much like those with normal lungs. If the pressure were positive the animal reduced it by forcing air out through the manometer. If negative, however, it slowly rises till the average is 0, but the animal will not allow it to become positive on inspiration if it can force the air through the tube. If it cannot (since the tube is closed) and the tube be opened after the animal is again quiet, the pressure will be found slightly lower. Since this occurs when there is no pulmonary wound, it need not be due to the forcing of air out through this. Hence pulmonary wounds have extremely little permeability for air, allowing a little to go slowly to pleura, but not in reverse direction, even by convulsive movements.

He now seeks the cause. The kind or extent of the wound had no influence; also he excludes closure of the wound by blood (see below), hence is convinced that the inherent elasticity of the lung, its anatomical structure, the direction and relations of its elastic fibers furnish this valve-like action, so that air cannot get from the pleura to the bronchi through the retracted parenchyma while there is but slow filtration in the reverse direction. This point was further tested by aspirating and injecting air through a tracheal canula of rabbits with these wounds in the lungs. Air could be forced in until the intrapleural pressure was $+3$ to $+4$ cm. H_2O, but no amount of aspirating would reduce the intrapleural pressure. A manometer was attached to the tracheal canula and the canula closed; in the dyspnœic movements that followed, the oscillations in the pleural and tracheal manometers were large, but those of the former greater, therefore in strong expiratory movements the expiratory pressure is higher in the pleura than in the trachea and in forced inspiration lower. Then on opening the canula of the trachea to relieve the animal, the intrapleural pressure is always found lowered, although no air had been expelled through the tracheal manometer. Hence strong expiratory movements reduce the intrapleural pressure.

VI. To eliminate wholly the obliteration of the wound by hæmorrhage, or by other vital factors, the work was repeated on dead rabbits, artificial respiration being performed, and similar results were obtained.

Isolated lungs, both rabbit and human, were tried, and the results only confirmed those of the above experiments. Neither air nor fluid could be drawn or forced through the lung from the pleura to the bronchi, although it could easily pass in the reverse direction, and in the latter case the counter force which the lung exerted (its elastic tension) was found to be 2 cm. H_2O. Similar results were obtained on the cadaver.

VII. Bard's experiments have, he says, confirmed all his clinical findings, and have demonstrated a new (?) fact concerning the lung. It is impossible in the normal lung to produce a wound permeable in both directions. If the fistula be open the case is always a valvular pneumothorax. The parenchyma of the lung itself acts as a valve always opening broncho-pleural-ward. It is a normal and necessary type of the pulmonary wounds, offering an insurmountable obstacle to air or fluid in the opposite direction, *unless adhesions prevent the collapse* of the lung *or the fistula has stiff walls.* The case cited of a wound 9 cm. deep shows that there

could be a large superficial cavity and still the pneumothorax be valvular if there was a zone of normal tissue between the cavity and the large bronchi, and in the immense majority of cases this is true. It is also true of other extensive cortical lesions, e. g., gangrene of the pleura, which does not form a cavity, despite extensive destruction of the lung.

The nomenclature of pneumothorax must be revised. Valvular is the ordinary usual form. Positive pressure with inspiration as well as expiration means an open fistula, and its absence, the obliteration of the fistula. The mechanism producing this positive pressure shows that this cannot cause the suffocating symptoms of the onset, and renders invalid the treatment proposed to check it; that is, cough sedatives and a permanent canula. The latter would be an injury, since it allows the passage of infected fluid through the pulmonary wound, which it also tends to keep open. The positive pressure when it has reached the level fixed for that patient, defends the pleura, obliterates the fistula and leaves compressed and at rest the causative lesion. This positive pressure is a beneficial adaptation and its formation should be aided. The violent suffocation at the onset of pneumothorax may be explained by the slowness of its development, and Bard proposes at his next opportunity to inject air into the pleura rather than aspirating it to relieve this symptom.

Conclusions.—1. Owing to the structure of the lung, wounds of its parenchyma of whatever kind or extent are permeable to gases and liquids only in the broncho-pleural direction. Only in some extensive lesions or when some obstacle to the retraction of the lung is present, such as occur in partial pneumothorax can gas or fluid pass in the pleuro-bronchial direction.

2. In general pneumothorax when the fistula is not obliterated the tension of the gas is positive in both phases of quiet respiration. This positive pressure results from the spontaneous tendency to equilibration of the intrapleural and intrabronchial gas, by the regular play of the respiratory movements without any aid by coughing. The so-called valvular pneumothorax is the normal form of general pneumothorax with open fistula. Coughing, etc., act more energetically on the intrapleural than the intrabronchial gas, hence they compress and make more atelectatic the lung, and push over the mediastinum, but do not cause any more air to enter the pleura through the pulmonary wound. The degree of this positive intra-pulmonary pressure is slight, is nearly constant in the same case, and shows respiratory oscillations of a few centimeters about a mean level of 6 to 8 cm. H_2O. After accidental disturbances it tends to gradually reestablish by quiet respiratory movements the former level.

3. The degree of this pressure is otherwise equal to and produced by the elasticity of the lung, which combined with the action of the thoracic wall creates a negative pressure when the lung is intact, but in pneumothorax with persisting wound, a positive pressure.

4. This positive pressure is an adaptation of pathological compensation which tends to close the wound and should be aided and not hindered at the onset of the trouble.

5. The measurement of the intrapleural pressure is necessary to determine the patency of the fistula. But one must consider not the average but the extreme pressures. If the fistula is still open the pressure will be positive during both phases of respiration. In partial pneumothorax (in which the fistula cannot close) it is positive at expiration and negative at inspiration. If the fistula can no longer open it will soon be negative at both phases of respiration.

342. Bard. Procédé simple pour mesurer le volume de la cavité d'un pneumothorax. *La Semaine médicale*, XXI, p. 337, 1901.

The method consists in a calculation based on the change of pressure before and after the aspiration of a known volume of gas.

343. Galliard. Pneumothorax suffocant traité par la pleurotomie. *Bull. et mém. de la Soc. méd. des Hôp. de Paris*, 3s., XVIII, p. 6, 1901.

A woman, 25 yrs. old, with partial and pure (no fluid) pneumothorax of the suffocative variety, with fetid expectoration, was operated upon, pleurotomy being performed. There was no infection of the pleura, the expectoration and the pulmonary disease ceased; she was discharged well. The cause may have been the expiratory efforts of the recent labor, three days previous, but the obstetricians and text-books he consulted state they had never seen that complication. The cyanosed suffocated condition of the patient indicated operative interference—respirations were 64 to the minute—and the result was marvellous.

344. Letulle. Pneumothorax par bronchiectasie chez un Diabétique. *Bull. et mém. de la Soc. méd. des Hôp. de Paris*, 3 s., XVIII, p. 690, 1901.

The writer comments on the rarity of pneumothorax in diabetes, although tuberculosis is so common in that disease.

The patient was a man 52 yrs. old, admitted, the writer thinks, with diabetes and pneumothorax, which latter condition autopsy showed was due to a small caseous area in the wall of a bronchiectatic cavity. The case is considered rare and is well described. It is the same case reported by Gébrak, 345.

345. Gébrak. Le pneumothorax dans la bronchiectasie. Paris, 1901.

The conclusions of this writer are:

1. In medical literature are but ten cases of pneumothorax due to bronchiectasis.

2. One such case is here reported due to the caseation of the tissue over the bronchial aneurism.

3. There may be one or several points of rupture.

4. The perforation may be small or as large as the end of the little finger.

The case here reported was of a diabetic, hence very rare, although in diabetes tuberculosis is very common, and is the same case Letulle reported.

346. **Hodenpyl.** Pneumothorax. *Proceedings of the New York Path. Soc.* (1899-1900), 1901, p. 181.

The patient was a gripman who was seized with severe pain in the chest while running his car. He died in a few hours of right-sided pneumothorax. There was advanced tuberculosis in both lungs. The displacement of the liver was extreme.

347. **Dalagénière.** Du pneumothorax chirurgical; ses dangers et sa valeur au point de vue de la chirurgie pleuropulmonaire d'après six observations. *Arch. prov. de chirurg.*, Dec., 1901.

The study of six cases led the writer to the following conclusions:

1. Pneumothorax produced rapidly is dangerous but not fatal, hence should be slowly produced. It is a valuable means of arresting certain cases of pulmonary hæmorrhage. Once produced it is innocuous.

2. The formation of pneumothorax can be arrested by suturing the lung in the wound.

3. Subsequent aspiration of air will relieve the dyspnœa, etc.

4. The surgeon may slowly produce in a sound pleura a pneumothorax, then open the pleura widely, explore the lung, suture in the wound the part needing operation, and then remove the air.

The operation was performed for severe hæmoptysis following a bullet wound; osteosarcoma of the sternum, intrapleural tumors, abscess of the lung, and other conditions.

348. **Hellin.** Ueber das Collabiren der Lunge beim Pneumothorax. *Berliner klinische Wochenschr.*, Oct. 7, 1901.

The question whether in pneumothorax that lung collapsed entirely or still received air from the trachea is still open. He cited seven different opinions on that point. The author will test it as follows: If rabbits with double pneumothorax can be kept alive, this will prove that the lungs do not entirely collapse.

Experiment 1.—Rabbits were used, double pneumothorax being produced by putting canulas of various sizes into the two sides of the chest. After one hour the wounds were closed. With a 5 mm. canula in each chest animals whose trachea was not over 5 mm. survived.

Exp. 2.—Wide openings were made in one pleura and closed in 1 to 2 min. and a similar operation at once performed on the other side. Several animals survived.

Exp. 3.—Wide openings (2 ribs being resected) were made in both sides at once and held open ½ to 1 min., then closed. The animals lived ½ to 1½ hours.

Exp. 4.—Wider opening of both chests simultaneously; closed. Animals lived 5 to 6 min.

Exp. 5.—Extirpation of right lung. In 6 days the left chest opened first with a fine canula, then by resection of two ribs, the wound held open 5 min., then closed. The animal died 6 wks. later.

The above experiments prove that after the finest or widest wounds of the

chest the lung does not perfectly collapse, since the animal does not at once die if double pneumothorax be produced. The lung does not collapse perfectly even when the pleural opening is as large as the trachea.

Concerning the re-expansion of the lung, he cites many opinions, but is sure Unverricht's is correct, i. e., the absorption of the air.

The author has shown that rabbits can survive a double pneumothorax; no complete double pneumothorax in men have yet been observed, all the cases described having adherent pleuræ.

349. Buchanan. Pneumothorax. *The Liverpool Medico-Chirurg. Journal*, XXII, p. 74, 1902.

A case of pneumothorax due to muscle strain in a healthy man. No fluid was present. The radiographs were very satisfactory. (Only a note given of the case.)

350. Fussell and Riesman. Spontaneous Non-tuberculous Pneumothorax. *Amer. Jour. of the Med. Sc.*, Aug., 1902.

Two cases are reported, which the writers believe were not tuberculous (although the tuberculin test was not tried).

The writers have collected 56 such cases and have carefully analyzed them, and arranged them in a very convenient table.

Their conclusions are:

1. Spontaneous non-tuberculous (?) pneumothorax occurs in healthy individuals. It is rare. It is most common in young men.

2. The pneumothorax is simple, i. e., there is no formation of fluid.

3. There is rarely any febrile reaction, except, perhaps, at the very beginning.

4. There is probably a moderate amount of emphysema in the lung which is not recognized during life and is not incompatible with health.

5. Aspiration is a certain and safe means of relief and should be resorted to in severe or prolonged attacks.

6. This form of pneumothorax is benign. All but one of the reported cases recovered, and in only one was there a fluid exudate.

351. Jochmann. A Peculiar Case of Pneumothorax. *Zeitschr. für klin. Med.*, 1902, p. 97.

The case is peculiar because of the absence of subjective symptoms, its remarkable course. Much aid was given by the skiagrams in the diagnosis and in following the course.

No cause could be assigned. There were no symptoms of tuberculosis, no history of injury. It must be included among those cases in which a lung-pleural fistula arises and then closes. In this case it must have opened a second time. He had almost no subjective symptoms and wished to continue his work. The skiagram was here an aid in diagnosis and in the study of the healing of the case.

352. Struppler. Pneumothorax Acutissimus in Incarcerated Diaphragmatic Hernia. *Münchener med. Wochenschr.*, April 15, 1902.

Struppler mentions the causes for pneumothorax on the side of the abdomen, also the possibility of zymotic pneumothorax. His is an extremely rare case of incarcerated diaphragmatic hernia with local peritonitis of the strangulated loop of intestine which infected the pleura, caused empyema, and the growth of gas-producing bacteria in the exudate causing pneumothorax without perforation of either the pleura or intestine. The development of the pneumothorax was very rapid and death occurred ten hours later.

353. Zuppinger. Pneumothorax im Kindesalter. *Wiener klin. Wochenschr.*, Jan. 2, 1902.

Girl, 2½ yrs. old, healthy, developed total right-sided pneumothorax, evidently during sleep, and died in thirty-six hours. At autopsy in the right pleura was found about 500 cc. of purulent fluid. The perforation was caused by a grain of corn 4 cm. long which projected from the lung into the pleura. The source is supposed to have been the mattress made of such material, which covered the child in bed. The covering of this was threadbare and this small grain of corn fell into the mouth and was aspirated.

The writer comments on the frequency of foreign bodies in the respiratory passages of children and cites cases in which these have led to pneumothorax; also to perforations from the stomach, etc. Also pneumothorax as a result of disease is discussed; gangrene is a more common cause than in adult life. In that hospital about 1 per cent of the cases of lung tuberculosis developed pneumothorax (two cases). Both of these girls owed their early death to measles, which in consumption predisposes to pneumothorax. He reports but three cases of gangrene of the lungs, one leading to pneumothorax, which he reports at length. It was a 26-mos. child with diphtheria, which died. At autopsy pyopneumothorax of the left side, gangrene of the inferior lobe, and thrombosis of the pulmonary artery were found. He mentions cases due to emphysema following whooping-cough, and other important occurrences.

This excellent paper should be consulted by all interested in pneumothorax of children since many references are given to articles not easily examined, and these articles are briefly reviewed.

354. Budingen. The Thorax Pressure-manometer and the New Lung Test. *Münch. med. Wochschr.*, June 3, 1902.

The author describes his trocar and canula, which will not injure the lung and admits of attachment to a manometer, which he has formerly published. His present purpose, however, is to defend his rights against Placzek, who has proposed an instrument for similar use.

355. Morris. A Remarkable Case of Pneumothorax. *Lancet*, Oct. 25, 1902.

Morris reports a case of a healthy man who fell from a window a distance of about 20 feet. Twenty minutes later he had recovered consciousness and

was complaining of great pain in the left chest. He found the greatest comfort in the knee-elbow posture. Traumatic emphysema was present over the left side, extending from the clavicle to the costal margin. There was no sign of external injury. Left-sided pneumothorax was present. No fractured rib could be found. On the day following the accident an extraordinary sound was heard in the chest synchronous with the cardiac systole. It was something between a click and the noise made by a bursting bubble and was distinctly heard all over the room. On auscultation in addition, a loud to-and-fro churning sound was heard over the præcordium. This clicking sound was audible to those standing by the bedside for four days and was always worse when he turned to his left side. The sounds are explained by the writer by the squeezing of bubbles of air between the pericardium and the parietal layers of the pleura. He considers pericarditis present. A fortnight later the man was pronounced well. The writer calls attention (1) to the curious nature of the accident; (2) the extraordinary rapidity with which the man got well, and (3) the exocardiac sounds, which he has not seen described in connection with any other case.

356. O'Neill. A Remarkable Case of Pneumothorax. *Lancet*, Nov. 8, 1902.

A few remarks on Mr. Morris' case. The writer considers rupture of the lungs without fracture of the rib rare, and due to the excessive inflation of the lungs owing to the deep inspiration made instinctively by the patient in his endeavor to avoid falling.

357. Maillart et Lasserre. Un cas de pneumothorax droit spontané; Examens radioscopiques et mensuration de la pression pleurale. *Revue méd. de la Suisse Romande*, Nov. 20, 1902.

The patient, 29 yrs. old, of excellent health, on the morning of his admission had developed, while stooping over, a sudden pneumothorax with very severe onset; he could not move to lie down or stand erect; he was speechless. That day was found exaggerated resonance on the right side, absence of fremitus. The breath sounds were replaced by an amphoric sound, distant, which resembled the sound of a bell through the arches of a cathedral. Coarse metallic râles were heard and beautiful metallic tinkling. The coin sound was intense; there was no succussion. The apex beat was 3 cm. outside the nipple line, the liver entirely below the costal margin reaching the umbilicus. The X-ray picture of the case is an excellent one. On the 7th day after onset (the distension and displacement of the heart and liver having diminished) the pressure at first was 0, and after the aspiration of air remained unchanged. The further notes are very complete. He left the hospital before entirely well and returned with some effusion. Radioscopy demonstrated in this case the absence of fluid, also of adhesions at the apex.

The "paradoxical respiratory movements of the diaphragm" were well seen in this case; that is, instead of its dome depressing during inspiration it rises, and lowers somewhat during expiration. This shows the diaphragm

to be paralyzed and to follow passively the movements of the abdomen. This is hard to explain (he gives four preceding references). It is a phenomenon present in open and closed pneumothorax but which disappears as fluid increases in the pleura. These movements are the reverse in pneumothorax from what they are in hydrothorax. Radioscopy also shows (Mignon) that the paralysis extends to the other muscles of respiration, hence the side is almost immobile. All the advantages of the Röntgen rays are discussed at length.

The determination of intrapleural tension in this case proved the presence of an open fistula (?).

They then collected all the cases they could find (51 in number) of similar nature, of which 16 were between the ages of 14 and 20 years; 45 were men. In 28 cases was there a history of no previous effort, in 22 such was stated to have preceded. The mortality was 10 per cent.

The duration was 10 days to 4 months, and in but three was an effusion present. Seven cases recurred.

This paper was published under Bard's supervision. The literary review is excellent.

358. Aron. Die Mechanik und Therapie des Pneumothorax. 1902.

With the absence of a historical review the author begins with:

I. *The intrapleural pressure in living persons.*—He reviews the work on animals, calls attention to the fact that similar observations had been unsatisfactorily made on patients, then proceeds to describe his method, using a glycerine manometer, in connection with the Bülan siphon drain. He discusses curves obtained in this way, the intrapleural pressure during inspiration and expiration in the lying and sitting posture, the length of each phase of respiration, etc. The patient was a woman who had been operated on for empyema. When lying, inspiration — 4 mm. Hg. was the average minimum pressure, and 1.28 sec. the average time; expiration, — 1.9 mm. Hg. the average maximum tension and 1.79 sec. the average time. Sitting up in bed the figures were: insp., — 4.52 mm., 1.07 sec.; exp., — 2.5 mm. and 1.76 sec. Sitting on a chair: insp., — 5.1 mm., 1.16 sec.; exp., — 3 mm., 1.54 sec. Sitting on a chair with the lung expanded as much as possible: insp., — 6.85 mm., 1.25 sec.; exp., — 3.9 mm. and 1.37 sec. The effects of coughing, laughing, etc., are well shown on a chart. The explanation for the discrepancy between his figures and Donder's is due, he thinks, to abnormal condition of his patient's lungs. Donders found the intrapleural tension in the human corpse to average — 6 mm. Hg. Aron found in this patient from — 4.5 to — 6.85 mm. Hg. for inspiration, — 1.9 to — 3.9 mm. for expiration.

II. *The intrapleural tension in living sound men.*—He found a normal person who was willing to have such determinations made. The technic is described, the harmlessness insisted upon. In 36 determinations he got as average tension at the height of quiet inspiration — 4.64 mm. Hg.; at the height of quiet expiration — 3.02 mm. Hg. (All these readings were

made with a glycerine manometer, but the corresponding values of a mercury one are given.) For inspiration the maximum reading was — 5.09 mm., the minimum — 4.23 mm., while the smallest value in expiration was — 3.29 mm. and the highest — 2.54 mm. Hg. This applies only to quiet respiration.

III. *Experimental studies of Pneumothorax.*—Aron recognizes Weil's work as classical, but because of its incompleteness he proposes to fill some of its spaces. He proposes to study the intrapleural condition at the moment pneumothorax arises, and as one form changes to another. He introduced a glycerine manometer into each pleural cavity and a mercury one into the carotid artery. These traced their curves on a revolving drum. His differ from Weil's methods since after trying various schemes he produced a valvular pneumothorax by injecting with a syringe at stated intervals a certain amount (10 ccm.) of air. He could thus study all three forms on the same animal, open pneumothorax by leaving the canula open, closed by occluding it, and valvular by repeated injections of 10 cc. of air. Thirty rabbits were experimented on and of these a complete series of observations obtained with nine.

When a pneumothorax is produced the respiratory mechanism must change, since one lung is rendered entirely functionless. Compensation might be attained by the respiration becoming more frequent (the most common way in rabbits) or deeper, or both, and his cases illustrated the employment of these three possible methods. As in animals so also in men one should recognize individual peculiarities in meeting the pathological condition.

Some of his results were the following:

After the production of a slight right-sided pneumothorax, or at the onset of a valvular one (i. e., after injection of 10 cc. of air), the respiration becomes more frequent (35½ to 37½ per min.) and more superficial (1.54 to 1.51 mm. Hg.). The excursion of the sound lung becomes smaller, while the intrapleural pressure rises (from — 2.73 mm. Hg. on insp. and — 1.19 on exp. to — 2.91 mm. Hg. on insp. and — 1.40 on exp.). Hence the lung is expanded more forcibly; the diaphragm must therefore descend lower. On the right side, however, the intrapleural pressure is diminished. After further injections the sound side continues to respond by deeper excursions until a certain point is reached, then the excursions become smaller and the animal compensates by more rapid respiration.

The result of further introductions of air into the right side (10 cc. each time) is that the tension on the left side gradually falls, while the expiratory tension, after a transitory fall, rises. Hence the sound lung does not expand as much as before, while expiration becomes less complete. The respiration on this side becomes, therefore, more superficial, after the transitory deepening, and more rapid. After 60 cc. had been injected respiration became slower, showing that the reserve strength for respiration was exhausted and death imminent. Hence at about this point the canula was opened and an *open pneumothorax* produced. The negative pressures during both inspiration and expiration were at once dimin-

ished (insp., — 2.59 to — 1.84; exp., — 1.46 to — 0.59 mm. Hg.), the depth of respiration became less, the rate faster.

Closing the canula now gives *closed pneumothorax.* On the sound side the negative pressure increases somewhat for inspiration and expiration, the respirations deepen, the rate sinks.

On the pneumothorax side (the right) meanwhile the negative tension steadily diminishes as the lung collapses. If the canula bore during open pneumothorax be small the right lung can take some part in respiration and the pressure in that pleura be slightly negative.

If the canula now be closed the right lung at once begins to share in respiration. The inspiratory pressure becomes negative, the expiratory remains positive.

The blood pressure rises, as air is injected, from 9.38 to 12.63 cm. Hg. In open pneumothorax it falls to 11.79 and remains, if the closed fistula be unchanged. The blood-pressure curve becomes irregular after considerable air has been injected. In conclusion he states it proven that it is impossible to change the intrapleural pressure of even one side without affecting the blood pressure and the intrapleural of the other side.

IV. *The intrapleural pressure in valvular pneumothorax of man.*—The tension was determined in the chest of a patient before and after the exit of air. Then by clamping the aspiration tube air was found to enter the pleura through the pulmonary wound, and the tension rose, to fall again after more air was aspirated, but even with siphon drain it was not possible to bring the pressure to 0, since evidently more air entered the wound than left by the needle, hence a constant positive pressure persisted. The autopsy showed a perforation, closure of which seemed impossible since adhesions prevented the collapse of the lung. He considers this a case of valvular pneumothorax. Before aspirating the average pressure at the height of inspiration was + 7.93 mm. Hg.; exp., 10.48 mm. After as much air as possible was removed: insp., + 2.85; exp., + 3.39 mm. Hg. After closing the aspiration tube the pressure rose to + 2.42 mm. during insp. and + 6.65 mm. at expiration. Aron says he must assume that the fistula was valvular, allowing air to enter with each inspiration and preventing its escape at expiration. Since in valvular pneumothorax aspiration of air is without avail, since it at once recollects, Aron advises a permanent open drain to keep tension low, since high tension is dangerous. But one cannot hope for much in such cases, since valvular pneumothorax occurs usually in advanced consumptives. He considers that the danger of preventing the closure of the fistula by the drain is not to be considered compared with the more important benefit of keeping the pressure reduced. Valvular pneumothorax deserves especial study, since it is the commonest and the most dangerous form. Cures from permanent closure of the fistula and absorption of the gas are very rare, but perhaps would not be so if permanent drains to prevent high tension were used. True, the external drain may keep the pulmonary fistula open, but of two evils choose the lesser. The danger of high tension his experiments have beautifully proven, for a continued high pressure means certain and near death.

V. *The mechanics of closed pneumothorax.*—The curve of intrapleural pressure was obtained from a tuberculous case, who died that night, and the autopsy of whom showed a closed fistula. Before allowing air to escape the pressure was found (insp., + 0.4 mm. Hg.; exp., + 5.0 mm. Hg.), and only after air was removed did it become negative (insp., — 7.9 mm.; exp., + 1.4 mm. Hg.). To look at the curve would, he thinks, allow diagnosis of closed pneumothorax following valvular, for only in that way could the pressure at the height of inspiration be positive. This total rise of 14 mm. Hg. during quiet respiration, — 9 mm. normal, + 5 in this case at the end of expiration, must, he is sure, be of moment in its effect on the circulation. Since the pressure after aspiration remained low the fistula was closed. Manometrical observations, therefore, have important diagnostic and prognostic value.

VI. *The mechanics of open pneumothorax.*—The case was one with an external fistula, none in the lung. (The pneumothorax followed the operation for empyema.) Hence while making the readings this was closed pneumothorax, the manometer occluding the fistula. The pressure oscillated around 0, hence none of the dangers of high pressure were present. Surgeons consider pneumothorax harmless (because it is open). Many readings gave the following: pressure at inspiration averaged — 2.62 mm. Hg.; at expiration, + 1.01 mm. Air was driven out by coughing, after which the average of thirty reading during inspiration was — 6.69 mm.; expiration, — 2.79 mm. He recommends Bülan's siphon drain as the best therapeutic measure in such cases of open (external) pneumothorax.

VII. *The spontaneous healing of pneumothorax.*—If the fistula be closed, the air is absorbed. This occurs more rapidly if the intrapleural pressure be just about zero than when higher. In the case of rabbits with sound lungs and pleura after the production of pneumothorax the negative pressure will be restored in seven hours, but if the pressure be raised to a positive one, so that that lung breathes less, restoration requires a few hours more. In men, however, it requires more time. He reports the case of an emphysematous man who as a result of inhaling hydrochloric acid fumes developed pneumothorax; there was no reason to suspect tuberculosis; the gas was absorbed four weeks later. Three manometric curves were obtained during this time. This man's condition troubled him very little, hence the severe dyspnœa and subjective symptoms which are the rule, Aron thinks depend much on individual peculiarities.

This long abstract of Aron's book is given, since it is the last careful study of the subject, the clinical observations are perhaps more complete than any previous, but especially because the author represents one group of workers who following Weil insist upon the danger of elevated intrapleural tension the result of valvular pneumothorax, and base their opinion on animal experiments. Bard, on the other hand, could not have asked for a better series of observations as evidence in favor of his views, and from them would have drawn diametrically opposite conclusions.

CHAPTER II.

The History of Pneumothorax.

To trace the development of our knowledge of the condition commonly termed pneumothorax is of greater interest than is the case of many commoner diseases. The history of our knowledge of a disease is at the same time the history of the growth of those sciences which have furnished the necessary data that the disease may be understood, hence few can claim an antiquity of more than a few decades. To understand pneumothorax on the other hand the observer need not be wise in bacteriology, chemistry or pathology, but only skilful in physical diagnosis. Hence many good observers of old have noted the presence and some of the symptoms and signs of this trouble and even given good explanations for them. It is true that pneumothorax has been understood but since the beginning of the nineteenth century. As a condition, however, it was well known at the middle of this era.

The date at which the succussion splash was first heard "is shrouded," to use Piorry's [42] term, "in the night of time." The writers of the complete works of Hippocrates [1] give an excellent description of it. To them it was the best diagnostic sign of empyema and was also important as a therapeutic measure. Although the name "Hippocratic" succussion is that commonly used, yet it is interesting that the complete works of Hippocrates have suffered almost as badly as the Pentateuch, and the higher critics of medicine will not admit that one of the passages in which this succussion is mentioned was written by Hippocrates. From the date of these writings until Laennec, about 2200 years, this physical sign was not held in very high repute. It was recognized, because some cases are on record in which it was heard across the room from the movements of the patient alone. It was probably often tried, but since the writers of the Hippocratic books had considered it a sign of empyema and since empyema is a common disease, while pneumothorax is a rare one, its value was very soon discredited and Laennec [41] states that not one of the followers of Hippocrates considered it at all. Morgagni,[18] in 1760, speaks quite feelingly on this point. "If it did occur in all cases of empyema," said he, "we would need no other sign. Although it sometimes is present, there are those who had never heard it and claim

that it never is present. It is heard only when a certain amount of fluid is present in the chest." He thinks that the physician is not justified in using it. The reason why the true value of this sign was not earlier recognized, says Houghton,[65] is the mistaken idea that air was normally present in the pleural cavity. This idea is attributed to Galen, and Morgagni in his argument concerning the value of the splash speaks as if he accepted it also. The falsity of this notion, however, fourteen years before Morgagni's publication had been proven by Haller.[14] Another reason why pneumothorax was not earlier understood was furnished by the cases reported of hernia of the lung. It would indeed be hard for a man to believe that there was a constant tendency to pneumothorax in the chest from the collapse of an elastic lung when after a wide wound the lung protruded itself and the opening had to be enlarged in order that it might be pushed back. Air, however, was found to issue from the chest by pathologists. Littré's [9] case is one of particular interest, while Boerhaave's [11] case of the famous Dutch admiral who ruptured his œsophagus by vomiting has become famous. Meckel,[17] in 1759, found the liver of one case in the right iliac fossa and the diaphragm projecting convexly into the abdominal cavity, but on puncturing the latter found that only air escaped. He said that this case alone was sufficient to refute the argument of the normal presence of air in the pleural cavity, and suggested that its presence there interfered with respiration by compressing the lung. The candle test seems also to have been used in some cases at the autopsy table to prove whether air did issue from the chest or not. It is of interest, however, that if the candle was held against a current of the inflammable gas which sometimes occurs in the chest, no record of it was made.

The surgeons also of this time discovered several cases of pneumothorax. Riolan [5] stated that on several occasions when operating for empyema only gas escaped. Combalusier [16] mentioned such a case and Ponteau [31] another. Nearly one hundred years before this in 1692, however, Purmann [4] mentioned wounds of the chest as if the occurrence were widely understood, stating that military surgeons understood well that no danger would result from air entering the pleural cavity and hence that the fear of this was no argument against the empyema operation. In 1732 Marequest de la Motte [13] made one statement, which is most interesting concerning the value of aspiration of perforating wounds of the chest, stating that at duels professionals were

always present to suck the wounds, and the success of this method for chest wounds was so well recognized that the good results were attributed to the devil. In 1760, Alexander Monro [21] proposed the empyema operation for cases with air in the pleural cavity and following him three or four (Kellie, Gooch, Richter), state that they used it with success. If it is true that they operated purposely to remove air in the pleural cavity it is hard to understand why Laennec should claim to be the first to recognize this condition in the living person.

The occurrence of pneumothorax in cases of fractured ribs is of particular interest. It may be that Celsus modified the Hippocratic treatment of empyema following fractured ribs because of the conditions produced by the presence also of air there. The first mention of subcutaneous empyema associated with fractured ribs is by Paré.[3] Several cases which may have been associated with pneumothorax were reported, but that of William Hunter,[23] in 1762, is to us of particular interest because of the fruit it bore. Following the publication of this case, four others were at once published and Hewson,[24] in 1762, then a young anatomical demonstrator, in a paper presented by Hunter, called attention to a fact which had escaped the latter's notice, that the symptoms in these cases were probably due to air within the pleural cavity. The few experiments that he performed upon animals and his one excellent case with autopsy may perhaps be considered as a starting point of our knowledge of pneumothorax for he also made the suggestion that the symptoms sometimes present in tuberculosis of the lungs may be due to the ulceration of the lung and the escape of air into the pleural cavity, although he knows of no case to substantiate this view. Benjamin Bell,[34] a surgeon, soon after this classified the cases which show this condition into: those following trauma, those due to putrefaction within the pleural cavity, and erosion of the lung. Bromfield [33] at this same time was interested in the subject and claims part of the credit for Hewson's discovery which seems to have attracted considerable attention at that time. These men, however, did not understand well the office of the elasticity of the lung. They considered that respiration was in some part due to the active participation of the lung itself, and this view would be well supported by cases of hernia of the lung and also the fact that the lung expanded best in those cases in which the external opening was the largest. It was John Bell who first corrected these views and

gave an excellent description of the mechanism producing pneumothorax. We have thus seen that at this time the surgeons understood well the condition which we now call pneumothorax. They expected it to be present in perforating wounds of the chest and suggested that it might be due to disease of the lungs and to putrefaction in the chest cavity. Warme,[51] in 1826, a French student who chose as subject for his thesis the wounds of the chest, gives an excellent review of the physiology of respiration and the office of the elasticity of the lung but says nothing of pneumothorax or of the epoch-making work of Laennec, although Laennec's publication had preceded him by seven years and he was probably one of his students. This would indicate that pneumothorax when first studied clinically was thought to be a separate condition from that which the surgeons had described.

Our knowledge of pneumothorax is usually stated to date from the thesis of Itard.[36] This French student reported five cases of tuberculosis of the lungs with pneumothorax. He gave a classification which is now in use, discussed the clinical symptoms and the differential diagnosis, and considered that the recognition of this condition will be of importance to the profession. None of these cases, however, were recognized during life and even Bayle[37] who wrote soon after this also failed to recognize them clinically. Itard's great contribution perhaps was the fact that he gave the condition the name which it now bears, thus giving it the individuality which it had hitherto lacked. At about this time the physicists had been making interesting discoveries concerning the nature of gas and its diffusibility. The work of Dalton concerning the intimate relation between gases and liquids and of Mitchell and Graham on their diffusibility are particularly to be mentioned. The influence of this new conception of the nature of gases very soon is felt in clinical medicine and Rullier[39] in 1815 in his article on empyema added air and gas to the list of effusions which may be present. The effusion of air in the chest he said may be due to wounds of the lungs, to ulceration of the lungs or to a foreign gas, and mentioned Itard's cases. He was skeptical, however, concerning Itard's explanation, although the latter was careful to state that he did not know their origin and refused to theorize concerning it, and stated that such cases may be due to a perverted secretion of the pleural membrane. Piorry[40] and Brierre de Boisment[47] at this time gave two very good reviews of the prevalent ideas of these conditions with gaseous exudates which at that time bore the name pneumatoses.

By far the most important contribution to pneumothorax is that of Laennec,[41] who claimed to be the first to recognize the condition during life. His contributions were the physical signs of pneumothorax, particularly those on auscultation, and rendered possible only by his invention of the stethoscope. It is not a trifle, however, that he showed for the first time the true meaning of the succussion splash and added that to the symptom-complex. His classification of cases is quite similar to that we now have, with the exception that he makes of considerable importance those groups due to decomposition within the pleural cavity, and to the abnormal secretion of the pleura. He was confident as were the others, that air could be secreted by the pericardium and by the arachnoid and synovial surfaces; this was accepted as a fact, why not then by the pleura? Piorry [42] writing the following year upon pneumothorax gives Itard and Laennec the entire credit of discovering this disease. At once cases were reported from all directions. Louis,[45] in 1825, republished some of his cases, changing the diagnosis to pneumothorax. Cases due to all possible conditions soon accumulated. In 1823, Davy [43] treated the condition experimentally, made the first gas analyses and demonstrated that air in the pleural cavity was not an irritating agency. Laennec considered that in the diagnosis of this condition auscultation was of prime importance, but Laennec was the discoverer of mediate auscultation. Louis considered that the clinical history was of great importance. Piorry,[54] the discoverer of mediate percussion, considers this of course the most important. Among other men whose names are to be particularly mentioned are those of Williams,[55] who in 1828 gave a very clear and satisfactory discussion of some of the signs; Skoda,[81] who also wrote on this subject and whose name is often used where Williams' deserves to be; Saussier,[78] who first compiled the cases then published; Trousseau,[117] who introduced the coin sound, and Weil,[165] whose experimental work was epoch-making.

It is of interest to note the part which the different countries have taken in the development of our knowledge of pneumothorax. It has, of course, been chiefly studied by the French. Physicians in Great Britain, however, very soon took up the subject, especially the clinicians in Dublin, who really gave us some of the best and earliest clinical descriptions of the cases. The Germans were long recognizing this condition. In 1833 appeared the first German article which we could find and this described one case. From 1840-1850, however,

eight German students chose it as the subject of their theses and in the next decade eleven. In this latter decade also appeared Skoda's work. From that time the contribution of Germany has been chiefly the discussion of the symptoms and animal experiments on the condition.

Since the time of Laennec the development of the subject has been along certain definite lines. The first of these was the discussion of the idiopathic cases. It is only to be expected that Laennec [41] should lay great emphasis on these and that his student, Venot,[45] should defend him in this position because these cases would be the group which first gave pneumothorax the right to its name. Belief in an idiopathic pneumothorax, that is a pneumothorax arising within the chest and in which the gas was not the atmospheric air, was very strong among the French. In Great Britain it early had firm believers and firm opponents and ever since then has found some to defend it. It is, however, the discussion of the physical signs which has ever and is now the point which attracts the greatest attention, and although we know much concerning the auscultatory signs of pneumothorax it may be said that we know scarcely anything more of their origin than did Laennec. Another group of these cases which has always been under dispute are those due to decomposition within the pleural cavity; either the decomposition of the lung as Itard suggested or the decomposition of the pleural fluid. That such cases should exist was early disputed and it is only about ten years ago that the opinion of Laennec was vindicated through the discovery of cases certainly due to the presence of gas-producing bacteria in the pleural exudate. The spontaneous cases have been recognized only since the middle of the 19th century and that is an ever-growing group. It is the presence of these cases which has changed so markedly the ideas concerning the prognosis of this disease. For the past twenty years pneumothorax has been chiefly discussed on its therapeutic side. At first the condition was studied only as a matter of interest and little was done for the patient; now it would seem that the consensus of opinion is to treat the case as if air were not present. If the air is under tension to allow it to escape through a hypodermic needle. If much fluid is present to remove that also. If the fluid be purulent, however, to treat the case as one of empyema.

CHAPTER III.

Etiology and Pathology, with the Clinical Histories of Cases.

Those who have collected large numbers of cases from the literature are Saussier [75] and Biach.[10] Saussier's compilation was of little value even at that time to show the relative frequency of the various forms, for even thus early only the unusual cases were reported. When Laennec, for example, reports one case and remarks that he gives this as illustration of some thirty similar ones that he has seen, of what value are percentages of sex, side, cause, etc., in which this group is reckoned as one case? Yet such compilations are of great value in searching for unusual features.

Much more can be learned from the reports of one clinic in which every case is included. Among these are those of Natrop,[179] 38 cases, and Bähr,[187] 40 cases (both from the Würzburg clinic), West, 101 cases; but the largest and most valuable, since it, as the experience of one man as well as of one clinic, is that of Drasche [316] who reported 198 cases.

I. PNEUMOTHORAX DUE TO DISEASES OF THE LUNG.

Pulmonary Tuberculosis.—Probably before Laennec, as since, this disease has caused the great majority of cases. With the exception of the possible reference to it in Hippocrates, the first distinct statement of the possibility is given by Hewson.[24] He does not know of any case in which the presence of air in the pleura was due to perforation of the lung by tuberculosis, but states it as a possibility. This was also the opinion of the surgeon Benjamin Bell.[34] With the demonstration by Itard [36] that pneumothorax was the result of tuberculosis begins the history of this condition as a medical disease, although Itard did not consider the condition the result of perforation of the lung but suspected that the gas arose by decomposition of a stagnant fluid. Laennec [41] first states clearly that the commonest cause of pneumothorax is the rupture of a tubercle or cavity into the pleura and bronchus, and that the gas was atmospheric air. Many cases of pneumothorax due to tuberculosis of the lungs were soon reported. The physicians of Dublin [65] were the first to give us some idea of the relative importance of this disease as a cause, and they ascribed nine-tenths of all cases to it. Saussier,[75] who col-

lected all of the cases reported to date, found that 81 of 147 were due to phthisis. Weil [180] reported 46 of 55 cases and these represented 3.8 per cent of the total tuberculosis material of that hospital; but reckoning on the basis of the ratio of fatal pneumothorax cases to fatal tuberculosis cases, a method of computation which he considers more correct than the former, he found pneumothorax present at autopsy in 10.1 per cent of the 355 cases of fatal phthisis. Biach [171] estimated that about 1 per cent of the consumptives treated in the Vienna hospitals had pneumothorax. For the fatal cases Chambers reported 3.7 per cent; Lebert, 5 per cent; Powell,[159] 5 per cent; West,[193] who reviews all of the above opinions, gives 5 per cent as his estimate of the fatal cases. He reported 101 new cases, 99 of which were certainly, and all probably, due to this disease. Galliard,[281] who used a more correct method of computation, determined that on one morning 1.054 per cent of all the phthisis cases in the hospitals of France had pneumothorax. Weil [180] reported 46 of 55 cases. Drasche,[316] who reported the largest individual series of cases (198), reported that these represented 2.36 per cent of the men, 1.13 per cent of the women who had been treated under his care for tuberculosis. He found the maximum occurred in the third decade.

Stage of the Disease.—It was the opinion of Weil [201] that acute tuberculosis was more favorable for the production of pneumothorax than was chronic, and stated that he had never seen pneumothorax in an afebrile case of tuberculosis. In explaining this the part played by pleural adhesions is of particular importance. Gairdner,[149] agreeing with Alison, considers that there is a tendency to the production of pneumothorax in every case of tuberculosis and that the reason for its non-production is the formation of adhesions over the cavity during its formation. This would explain the relative scarcity of perforations at the apex where cavity formation is the most common. This fact was noted by Houghton [65] and has been confirmed by almost every later writer, so that the discovery of a perforation at the apex entitles a case to a report (Stokes [70]). Wintrich [111] noted that it is the smaller tubercles which break down under the pleura before the formation of adhesions which explains many cases of pneumothorax and that these cavities are so small that they escape the physical examination. Desplats [158] reported a case in which a small subpleural tubercle had caused pneumothorax and without a trace of adhesions over it. This case, a rare one, died within

a few hours. The case of Thompson [75] is interesting in this particular, for he considered the coating of the pleura with false membrane prevented the formation of adhesions and hence the pleural cavity was not protected, and when this cavity broke down pneumothorax was the result. Because of the presence of these adhesions many cases of tuberculous pneumothorax are circumscribed, Galliard [261] noting the fact that total pneumothorax in this condition was rare. Duplant [325] stated that pneumothorax in tuberculosis was always partial at first and that later the lung gradually tears itself loose from the parietal pleura, which process can be followed clinically. In this connection Drasche's [316] cases of pneumothorax are interesting.

But if on the one hand the formation of adhesions is the process which protects most tuberculous cases from pneumothorax, on the other hand it is their presence which explains an ever-increasing number of cases, increasing because of their more general recognition. That many of the cases of so-called spontaneous pneumothorax, that is, pneumothorax developing in apparently normal individuals as the result sometimes of a strain, are due to the tearing of the lung by one of these adhesions is now generally admitted. The adhesion fixes one point of the lung to the parietal pleura and hence in a violent effort furnishes the necessary condition for a tear of the visceral pleura at this point. The recognition of this is attributed to Tripier (Duplant [325]). A further discussion of these cases will appear under the heading, "Spontaneous Pneumothorax." (See also Lees.[85])

Among the interesting cases of pneumothorax due to tuberculosis the following may be cited. Roe [130] reported a case of tuberculosis of the left lung which by perforation through the mediastinum caused pneumothorax of the right side. Russell's case [182] was both latent and early. The case of Coats (Inglis [170]) was one developing suddenly as the result of hard work, and presented subcutaneous emphysema in the mamillary region. The case of Vetlesen [177] is ascribed to tuberculosis. It is a case which ran a seven months' course without effusion, without fever and the patient feeling quite well. West [200] reports a case in which the patient died of suffocation, the contents of the tuberculous cavity being expressed into the bronchus by the compression of the collapsing lung. Lundie [285] reports one of spontaneous pneumothorax due to tuberculosis accompanied by pneumopericardium.

THE EFFECT OF PNEUMOTHORAX ON THE TUBERCULOUS PROCESS.—Houghton was the first we found to claim that the presence of pneumothorax had a favorable influence over the course of the tuberculosis. He reports one case which suggests this influence. From that time this subject has received an animated discussion; Stokes [70] denied it, also Saussier,[78] and West [297] stated emphatically that no such favorable influence could be attributed to pneumothorax. On the other hand, Hughes [107] reports the first case on record which might be called a cure, the pneumothorax resulting in the arrest of the disease of the one lung, the patient dying later of tuberculosis of the other lung. Another case is that of Banks.[110] The artificial production of pneumothorax as a therapeutic measure for tuberculosis has been considered for many years. Bruen [214] advised it. The method, however, usually goes under Potain's name who in 1888 reported favorable results in a few cases. Bernard [299] discussed the results of this method and although considering it dangerous to limit a person's respiratory mechanism to one lung, approves of its use in a few selected cases.

THE SITUATION OF THE PERFORATION was also of much interest. The apex of the lung, the most frequent seat of cavity formation, was such an infrequent seat of the perforation that Stokes [70] saw fit to mention one such case as rare. Houghton's conclusion was that the most frequent seat of perforation was the inferior part of the upper lobe, next came the superior part of the middle. In the other collections of cases this subject will be found still further discussed. The proximity to adhesions has been mentioned. At this point may be mentioned Bouchet's [300] case in which, strange to say, the lung was perforated through a very thick pleura.

THE SIDE AFFECTED.—Reynaud,[58] to settle this point, early collected all the cases he could find, and found in almost twice as many cases the perforation was of the left side. This was also Houghton's [65] opinion. In Saussier's review exactly the same relation was found. Weil [180] agreed and suggested that the reason might be that the most motile part of the lungs was the cardiac lapel on the left side. In Bähr's [187] cases 20, however, were of the right side and only 16 on the left, and of Drasche's [316] 198 cases 55 per cent were right-sided cases.

A similar question was also considered. Was it the MOST INVOLVED LUNG which perforated, and the consensus of opinion is that

it was the lung with the weakest spot on the periphery whatever the condition of its deeper tissues may be.

The Size of the Perforation varies within wide limits, sometimes too small to find except by inflation under water, sometimes 2 to 3 cm. in diameter (Saussier). Houghton [65] said that the perforation tended to increase in size, but rarely exceeded that of a shilling. As a rule the perforation is of a small cavity.

The number of perforations also varies; as a rule but one; there may be many (Saussier [78]).

A remarkable case is that of Roe.[130] The right lung was perforated, but through an adherent mediastinum, hence the pneumothorax was on the left side. As already noted, the perforation of the mediastinum may transform a single to a double pneumothorax.

The cause of the rupture in tuberculosis cases is not as often coughing as would be imagined. This seemed the cause in 34 of the 198 cases reported by Drasche.[316] Crowdy's [308] patient was merely turning over in bed. Delafield's was sitting quietly at the table.

Total tuberculous pneumothorax.

CASE 1.—*Tuberculous pyopneumothorax of the right side due to extensive cavity formation; open fistula; death; autopsy.*

John Sn., aged 46, white. **Medical Number 55.**

Admitted July 2, 1889.

Died July 27, 1889.

Diagnosis.—*Phthisis; pneumothorax.*

He complained of cough; sore throat; shortness of breath; much expectoration; loss of weight and strength.

Family history is negative; no tuberculosis.

Past History.—He was strong as a young man; habits have been good. He has had no illnesses.

Present illness began with weakness in the lower limbs and loss of appetite last fall. Cough began in the early winter. He lost flesh and strength gradually. The cough slowly increased until two months ago when he had to give up work, and has been somewhat worse for the past two weeks. No night sweats at any time. The cough is worse at night. He expectorates, after eating, white mucus; never blood. Thinks he has lost 25 pounds during the last six months. He has had sore throat for the past two weeks. At present the cough is not so bad as it was a few weeks ago. Shortness of breath is present on exertion. The appetite is poor. Temperature on admission 99.6; pulse 116.

Physical Examination.—The patient is pale; respiration quiet, 20 to the minute. The chest is well formed. Expansion is less on the right at the apex and on deep inspiration less at the right base. Vocal fremitus

is increased on the right. On percussion the right chest is dull in the infra-clavicular region and clear to the nipple though a bit impaired. There is dulness at the upper border of the 5th rib. In the back the resonance is markedly impaired in the infra-spinous, and especially the subscapular regions of the right side.

On auscultation the left apex is normal. On the right there is cavernous respiration to the upper margin of the 3rd rib. Splashing resonant râles heard here; exquisite pectoral râles and crackling râles are heard in the mamillary region and the lower axilla posteriorly. Distant cavernous sounds are heard at the apex; moist sounds at the scapular and infrascapular regions. "The vomica is evidently a large one involving a great part of the apex." There are a few moist râles in the apex posteriorly. The heart sounds are clear. The abdomen is negative.

The sputum showed both tubercle bacilli and elastic tissue.

July 18. The patient has been better for the past few days. Is still a little flighty at times. Pulse is 120. Immobility of the right side is much more marked. When he draws a deep breath the right side hardly moves at all. On percussion the right side anteriorly shows impaired resonance; at the right base posteriorly flat tympany. Breathing at the right apex and in the lower scapular region is amphoric, particularly on coughing. The râles are also amphoric. The bell sound is distinct. Metallic tinkling is very clear anteriorly. Crackling râles are also heard at the apex and base.

July 19. Pulse 120. Over the whole front of the right chest there is a full clear note, not tympanitic. On inspiration a rough harsh, not amphoric murmur at the end of which is a distinct metallic tinkling. The expiration is louder, rougher, but distinctly amphoric. Amphoric sounds are heard as low as the 7th rib. The liver is depressed. The right side of the chest is one-half inch larger in circumference than the left.

July 26. The condition is not altered materially since the last note. The patient has weakened gradually, is flighty most of the time.

July 27. The patient became weaker and died this day. The temperature has been very irregular, ranging from 98.5-103.5.

Autopsy No. 11.

Anatomical Diagnosis.—*Phthisis pulmonalis. Pneumothorax, right. Ulceration of intestine; perforation. Peritonitis. Ulceration of larynx. Adenoma suprarenal.*

Medium sized, emaciated man. Rigor mortis present. Nothing of note on skin.

Diaphragm corresponds to the 7th rib on right, 5th rib on left. On right side behind it is very much depressed in contrast to left.

A pipette in the 3rd right interspace in the region of pneumothorax; there was not pressure enough of air to disturb a flame.

The left lung covers anterior mediastinum but does not reach beyond the middle line. Right extreme pyopneumothorax; air sac extends anter-

iorly between sternum to middle line and above to the extreme apex in front; the finger at the edge of the scalenus touches the top of the cavity. The lower, that is the posterior 4th of the cavity is filled with creamy pus (500 cc.). The lung is compressed against the anterior mediastinum and spine.

Left lung is crepitant throughout. Many nodular tubercles can be felt. On the lateral aspect of the upper lobe is a thickened opaque patch on pleura which on section seems to be connected with a mass of old fibroid tubercles the size of a large walnut. The center of the mass is dark and fibroid, the periphery greyish with many miliary tubercles. Throughout both lobes are numerous recent patches of tubercles, some of which are undergoing caseation and several spots of softening have occurred.

Right lung.—Pleura is everywhere thickened and is covered everywhere with dense greyish-white exudate. The lung is nowhere crepitant. At the posterior part of the upper lobe there is a perforation, oval in shape, 10 x 7 mm., which leads directly into a series of cavities. Upper lobe, including apex and antero-lateral portions, is a solid caseous (on section) mass; posterior part of apex made up of recent cavity the size of a small orange. The perforation is in a small lateral off-shoot of the larger cavity. The tissue at the extreme apex is fibroid. The lower lobe contains a large group of caseous tubercles at the apex, and a few scattered nodules throughout the remainder of the condensed airless tissue. The middle lobe is airless except at extreme antero-lateral border.

The apex of the heart is directed a little more vertically than normal—almost in a line toward anterior spine of ilium. Distance from middle line at least 10 cm. The extreme right margin of heart (that is right auricle) is 5 cm. from middle line in 4th interspace. At least one-third of the anterior portion of the heart is made up of the left ventricle.

Liver is one hand's breadth below the costal margin.

This then is a case of right-sided pneumothorax developing in a chronic case of phthisis of from eight to ten months' duration. On admission there were the signs of cavity of the right apex, and while in the ward, even while the patient seemed improving, pneumothorax gradually supervened with evidently no marked symptoms at onset. He died about ten days later.

The disease of this lung was found to be extreme and the left lung also was extensively involved. This is interesting considering the latency of onset and the fact that the pneumothorax was total. The fistula was a large patent one opening into a series of cavities. During life the loud respiration was a feature, yet the amphoric quality was present only on expiration. Distension of the chest, hyperresonance, the coin sound and metallic tinkling were present. The distension of the chest and the displacement of the organs

were a striking feature of the case during life and on the autopsy table, yet the apparent patency of the fistula and the undisturbed flame suggest that these were not due to positive intrathoracic pressure.

The vertical position of the heart is worthy of mention.

CASE 2.—*Pneumothorax of the left side, due to phthisis; death.*

Jessie G., aged 24, white. **Medical Number 68.**

Admitted July 10, 1889.

Died August 24, 1889.

CLINICAL DIAGNOSIS.—*Phthisis pulmonalis, pyopneumothorax.*

The patient complained of cough, shortness of breath, loss of flesh and strength.

FAMILY HISTORY is negative.

She has had the diseases of childhood, malaria when 16 years old. Was strong and healthy as a girl. She never had to work until one year ago. She was then adopted by a lady who died of consumption later. The patient was with her a great deal for the two years she was ill, in the room with her all day and sometimes slept with her. She has had a cough for two years but did not notice it, feeling sure it was only an ordinary cold until six months ago when she took a cold after which the cough soon began and then she noticed that she was losing flesh and strength. This has continued gradually. The first expectoration was noticed six months ago. She continued work for three months although during this time she had frequent fainting spells from weakness following severe attacks of coughing. Shortness of breath has been noticeable during the past three weeks during which time she has been in bed most of the time. The night sweats have been severe for the past six months. She has had but one hæmorrhage, some time during last December when she expectorated about one cupful of blood. The voice has been husky for three weeks. The hearing has been affected for two weeks. The appetite has been very poor; the food causes pain in the stomach and a heavy feeling. She says the shortness of breath is her worst feature. Her cough keeps her awake.

PHYSICAL EXAMINATION.—The chest is very well formed; *expansion* is much more on the right than on the left; the left apex expands well. There is not that extreme immobility sometimes seen in pneumothorax. The clavicle on the right side is more prominent than on the left. The intercostal spaces on the right are obliterated. Percussion.—There is nowhere marked tympanitic resonance. The note at the right apex is higher pitched, approaching dulness. Resonance is defective in the left infraclavicular region. Vocal fremitus is not entirely absent but not so clear on the left as on the right. Posteriorly resonance is defective at both apices and on the left side as low as the angle of the scapula. There is nowhere tympany. At the base the tone is a little higher pitched. Auscultation.—Numerous moist râles are heard in the right infraclavicular region. Expiration is somewhat prolonged and hollow. The râles extend

along the right sternal margin to the 4th rib. On the left are most marked cavernous sounds to the 4th rib. Breathing is typically amphoric and the râles all of a very musical quality. The cracked pot sound is marked. Posteriorly there are cavity signs on the left side with large râles. Expiration is very hollow, not distinctly amphoric. All along the spine these large râles are heard almost to the angle of the scapula. Here there is most typical amphoric breathing with loud splashing amphoric râles. The bell sound is heard. Hippocratic succussion is not obtained. The right side by measurement is one-half inch larger than the left.

Heart.—There is neither impulse nor cardiac dulness to the left of the sternum. The sounds are loud and clear beneath the sternum. Tubercle bacilli and elastic tissue are present in the sputum. The patient gradually grew weaker until August 24th when she died. The temperature has been remarkably irregular, ranging from 97 to 103.

In this case of phthisis of two and a half years' duration the presence of pneumothorax of the left side was indicated by the amphoric respiration, the positive coin sound and the obliteration of cardiac dulness. It was perhaps pure.

CASE 3.—*Pyopneumothorax, right side, due to tuberculosis with extensive cavity formation; operation; death; autopsy.*

John R. M., 35 yrs. old, white. **Medical Number 433.**

Admitted March 18, 1890.

Transferred March 26, 1890.

Clinical Diagnosis.—*Cardio-valvular disease; Phthisis; Pyopneumothorax.*

Complained of "cough, expectoration, and persistent loss of flesh and strength."

Family history good.

Past History.—The only illness he had was diphtheria, fifteen years ago, since which time he has had laryngeal trouble. Was a blacksmith till 4 years ago when he became a bartender working at night, but drinks little.

Present Illness.—This began Jan. 3, with a heavy cold, a paroxysmal cough followed by vomiting; no chill, no fever; he went to bed and remained there eight or ten days; the cough continued and night sweats began. He returned to work for two nights, then he had pains all through the chest, with dyspnœa and cough which again sent him to bed where he has remained till admission.

The night sweats are profuse. He has lost thirty-five pounds weight, and much strength.

The sputum is mucopurulent, abundant, about 450 ccs. in twelve hours, and contains many tubercle bacilli.

The urine contains a trace of albumin.

Physical Examination.—The patient is emaciated and the face flushed. Pulse 108. Respiration 30.

Thorax.—The expansion is deficient at the upper right lobe and right base; no marked depression above the clavicles. On percussion there is dulness on the left side from the apex to the first interspace. Fremitus is increased as low as the third rib. The note is clear in the mamillary and axiliary regions. On the right side there is hyperresonance in front extending into the mid-axilla. Resonance extends to the upper border of the 8th rib in the mammillary line. The intercostal spaces are less marked on this side. From the mid-axillary line there is dulness almost amounting to flatness on percussion which is movable, disappearing when the patient is on the left side. There is marked Hippocratic succussion. The percussion behind in the erect posture is impaired at the right apex. Dulness in the interscapular region; hyperresonance in the lower scapular region; at the base beginning at the 7th rib, flatness. Fremitus is almost abolished in this lower zone.

Auscultation.—There is harsh breathing over the whole of the left lung. At the 4th interspace in front are sharp crackling râles; no tubular breathing. Right side. In front there is intense tubular breathing over the whole of the chest and as far as the mid-axilla in the recumbent posture. Behind in the erect posture the amphoric breathing extends as far as the angle of the scapula. Below this the breath sounds are absent. On succussion there is loud metallic splashing; no adventitious sounds are heard over the area of dulness.

Heart.—The impulse is wavy in the 3rd, 4th and 5th interspaces, and reaches two fingers' breadth outside the nipple line. There is here a short systolic thrill. The first sound is loud and rough accompanied by a distant murmur. At the aortic area the cardiac sounds are heard but are distant. There is a diastolic murmur at the base. The liver dulness in the right mammillary line extends from the 8th rib to 2½ inches below the costal margin. The spleen is not palpable. The abdomen is negative.

On March 26th he was transferred to the surgical side for operation.

Surg. No. 389.

Operation, March 27.

Portions of the 6th and 7th ribs in the anterior axillary line were resected, and the cavity drained. By estimation there was about a gallon of thin greenish-yellow pus present. The whole lung was apparently collapsed and could be neither seen nor felt.

The cavity was packed with gauze.

The patient improved temporarily but died April 2, 1890.

The temperature after the operation ranged from normal to 102.

Autopsy No. 82.

Anatomical Diagnosis.—*Operation wound on right side of chest (removal of portions of 5th and 6th ribs). Pneumothorax from perforation of tuberculous cavity. Dilatation of heart. Tuberculosis of both lungs. Syphilitic induration of testicles and cicatrix of penis.*

Body slightly built, 167 cm. long, pale, pupils equal and dilated. On the right side of body, commencing slightly posterior to middle axillary

line, at point opposite lower border of nipple, extending obliquely downward and forward, parallel with ribs, is an incised wound from which portions of the 5th and 6th ribs were removed. The wound is 7 cm. in length.

Diaphragm at right side is at lower border of 4th rib, on left side at 6th intercostal space.

Left lung is free from adhesions; in the pleural cavity is a small amount of bloody serum. Right lung is collapsed.

In the left lung on section there is found a small cavity in apex surrounded by tolerably firm caseous pneumonia. In the lower portion of upper lobe are numerous small ragged cavities and disseminated tubercles. The pleura is thickened immediately over the cavity.

Right lung is firm and compressed. Both parietal and visceral pleuræ are covered with thick caseous exudation. Lung is intimately adherent at upper and posterior portions of upper lobe and extreme lower anterior tip of lower lobe; elsewhere it is free. At lower portion of upper adhesion is a dense fibrous thickening of pleura. Immediately beneath this is a large cavity with apparently no lung tissue over it. The cavity communicates with the pleural sac just at the anterior edge of the adhesions. Here there is an opening 1 cm. in diameter. Elsewhere in lung there are disseminated tubercles and areas of caseation. Lung tissue is dense and firm. Nearly all the lung sinks in water. Bronchi are prominent.

The apex of the heart lies in mammillary line at the lower border of 6th rib.

Liver is 6 cm. below edges of ribs in axillary line; in median line 9 cm. below tip of cartilage.

In this case the phthisis was of about ten weeks' standing when the patient was admitted. When the perforation of the lung occurred there is no way of knowing, although it may have developed two months before, at the time the patient was at work. The size of the fistula indicates a very chronic, long-standing process. The physical signs were: scarcely any distension of the side, hyperresonance, movable dulness, the succussion splash, and amphoric breathing. The metallic tinkling was not noted. The heart and liver were displaced, the former taking a more vertical position. The patient died six days after the operation. The interesting feature at autopsy was the large patent fistula into a large cavity of an airless lung, at the anterior surface of the upper lobe.

CASE 4.—*Pyopneumothorax of the left side, due to cavity formation; death; autopsy.*

Lewelin Y., colored, 27 yrs. old. **Med. No. 536.**

Admitted May 16, 1890.

Died June 24, 1890.

Clinical Diagnosis.—*Phthisis Pulmonalis.*

Complaint.—Severe cough, large amount of sputum, slight pain in the chest, weakness.

Family History, negative, no tuberculosis.

Past History.—He has always been well, measles being the only illness till the present.

Present Illness.—This began about four weeks ago when he caught cold. He has had no chills but has had fever every night since. The cough and expectoration have been continuous. The sputum has been abundant but never blood stained. He has had no night sweats, no nausea or vomiting. His appetite has failed, he has lost much weight. The dyspnœa has been marked and there has been some pain in the chest.

Physical Examination.—The patient is emaciated. The fever is high, pulse and respiration (30) rapid. The left side of the chest expands very little. There is flattening in the left infraclavicular and mammary regions. The left clavicle is prominent.

Percussion.—The right side is clear throughout. On the left side there is a tympanitic note to the sixth rib in the axilla. In the 2nd interspace front is the "cracked pot note." Posteriorly there is dulness to the angle of the scapula. Vocal fremitus is increased in the subspinous region. Auscultation: there is loud, blowing breathing over the right front, tubular in the mammary region with fine râles. In the axilla is normal respiration. Posteriorly the breathing is feeble, no râles are heard.

On the left side is cavernous respiration to the 5th space in the axilla and almost to the angle of the scapula. The whispered voice is most intense. Liquid râles very numerous are heard to the base.

The apex beat of the heart is neither visible nor palpable, the dulness is quite limited in extent. The sounds are feeble, but the 2nd aortic is ringing. The sputum is mucopurulent, blood-streaked, nummular in character, and contains elastic tissue and tubercle bacilli in abundance.

The temperature ran an irregular course, for the most part between 99 to 103°, but on some occasions reaching 104 to 105°. The pulse was usually 100 to 120 and the respirations 24 to 40 to the minute. He died June 24, 1890.

Autopsy No. 105.

Anatomical Diagnosis.—*Tuberculosis of both lungs; complete caseation (tuberculous pneumonia) of left; pyopneumothorax, left. Atrophy of heart. Tuberculous ulcers in small intestine. Thrombosis in lower femoral and popliteal veins.*

Body 176 cm. long, emaciated, left lower extremity œdematous below knee, no rigor-mortis.

Diaphragm on right side is at the lower border of 4th rib; on left side, middle of 6th.

The left lung is apparently solid, retracted, occupying but small space in the thoracic cavity. In the left pleural cavity there are about 300 cc. of sero-purulent fluid. Right lung is firmly adherent.

The entire left lung sinks in water; weighs 1822 grammes. Great vessels at the root of the lung are free. The pleural surface is covered with fresh fibrinous exudate. Both lobes are tightly glued together. The upper lobe is of a general whitish grey color. Immediately beneath the pleura are innumerable small greyish-white points, especially on the anterior edge of lung. On the middle of anterior surface are large ecchymoses. In the pleural exudation, what appeared at first sight to be simply fibrinous exudate, proves to be a light membrane easily peeled off with innumerable blood-vessels from which the hæmorrhages had taken place. In the upper lobe in the posterior middle is a cavity communicating directly with the pleura. In the apex of the lung several small cavities communicating with each other are found. On section the lung shows through both lobes, but especially the upper, grey caseous consolidation over a large area. In the lung between these areas are miliary tubercles and gelatinous pneumonia. In the lower lobe the complete caseation is more toward the middle of the lung; on the outside and towards the base are innumerable masses of miliary tubercles.

Right lung is firmly adherent to pleura. On the pleural surface between the lobes are numerous miliary tubercles and ecchymoses. Large vessels at the root are free. Bronchi are dilated. There are large areas of consolidation scattered throughout the whole lung, this consolidation taking the form of areas of caseation with miliary tubercles between and around the areas. The lung tissue is in part entirely œdematous and in part in a stage of gelatinous pneumonia. This is especially noted in the upper lobe. In the lower lobe are smaller areas of caseation surrounded by œdematous lung tissue.

Liver entirely concealed, its lower border everywhere being 3 cm. above line of ribs.

In this case the tuberculosis was of about four weeks' duration, and very severe. There are no symptoms by which to date the onset of the pneumothorax, although it was a case of total pneumothorax, and unsuspected during life; his very ill condition may have prevented extended examination. The percussion note of the left chest was described as tympanitic, the cracked pot sound was present, the cardiac dulness diminished, the respiration was cavernous, which at least means that it was intense, and which is interesting since the fistula was found patent, opening into a cavity in the middle of the posterior surface of the upper lobe. The lung was evidently functionless.

CASE 5.—*Pyopneumothorax of the right side, total, due to extensive cavity formation; death; autopsy.*

Adolph H., white, 31 yrs. old. **Med. No. 1815.**

Admitted Jan. 5, 1892.

Died April 15, 1892.

Clinical Diagnosis.—*Pulmonary tuberculosis; pyopneumothorax.*

Complaint.—"Shortness of breath, and pain in the chest."

Family History.—His mother died of consumption.

Past History.—He has never been ill before. Has been a sailor.

Present Illness.—A cough began seven months ago, and became very violent. He was able to work until two weeks ago, when he had to take to his bed. For some time he has been losing appetite and growing weaker. He has vomited considerably. On the evening of January 1, after coughing a great deal, he fell asleep, but awoke feeling very short of breath. He has had no fever, no night sweats, and little expectoration till lately.

Physical Examination.—The patient is a healthy-looking man. There is no respiratory distress. The face is somewhat cyanotic. Pulse 118. respiration 38. Expiration is prolonged and somewhat wheezing.

Thorax.—Well formed; the left side expands a little more than the right. The intercostal spaces on the right axilla are less marked than on the left. The apex beat is felt in the 5th i. s., 1½ in. outside the nipple line.

Percussion.—The right side is relatively duller than the left. In the erect posture the note in the infraclavicular region is tympanitic; not while recumbent. Resonance extends to the costal margin in front. The tactile fremitus is diminished on the right side.

Auscultation.—Back; left side; inspiration loud and rough with an occasional râle; expiration prolonged. In the infrascapular region is heard a to-and-fro friction rub. Right side; in the suprascapular region is typical amphoric respiration with metallic tinkling; this is heard all down the back. At the extreme base is almost silence; the contrast between the two sides is extraordinary. The coin sound is well marked, particularly in the upper half of the chest.

Fronts.—Left side: respiration is everywhere breezy and loud. Right side: intense amphoric respiration in the infraclavicular and mammary regions.

Exploratory puncture in the 8th i. s. gave no fluid.

The urine is negative.

Jan. 8. The Hippocratic succussion is marked.

Jan. 11. The note on percussion (in the semi-recumbent position) is clear to the costal margin. At the posterior base is flat tympany. The movable dulness is marked. Everywhere is heard the most typical amphoric breathing. Metallic tinkling is heard in the middle of the back. Tubercle bacilli are found in the sputum.

Jan. 27. An acute pleurisy develops on the left side.

Feb. 1. The patient for the past three days has been walking about the ward. The physical signs are much the same. The patient has had severe coughing spells. Cardiac impulse is in the 4th and 5th i. s. below and outside the nipple. Expansion is still slight on the right side. Percussion on the right side is high pitched in the infraclavicu-

lar region, neither amphoric nor tympanitic. The note is rather full pitched in the 3rd and 4th spaces; it is resonant to the lower margin of the 5th rib. The sounds on coughing are amphoric. The breathing is not amphoric either in the mammary or infraclavicular region. Dulness begins behind about two fingers' breadth below the angle of the scapula. There are still excellent amphoric signs behind. Movable dulness is marked.

March 28. The right pleural cavity was aspirated at the 9th i. s. Twenty ounces of thin purulent fluid withdrawn.

April 4. The patient has become steadily weaker. The fluid has increased since the tapping. Early to-day the patient died.

The temperature during the three and a half months' stay was very irregular, ranging from 96 to 104° F.

Autopsy No. 294.

Anatomical Diagnosis.—*Tuberculous phthisis with cavities and caseation. Pyopneumothorax of right side with compression of lung and downward dislocation of liver from perforation of tuberculous cavity. Tuberculous ulcers of intestine.*

Body large, slightly emaciated, lower extremities œdematous. Extensive post-mortem congestion. Face somewhat cyanotic.

Diaphragm on the right side is bulged downward, fluctuating; the lower border is 4 cm. below level of ribs. The diaphragm on the left side is in the 7th intercostal space.

The entire right pleural cavity is filled with a large amount, more than 4000 cc., of tolerably thin, creamy pus and a small amount of air. The pleura extends far over on the left side, 5 cm. to the left of median line. The parietal and visceral pleuræ on the right side covered with a shaggy nap of fibrin and pus. There are some naps of the same material in the pleural fluid. The lung is compressed into a small mass at the top and posterior part of the cavity. It is dense and calcified and contains little or no air. In the apex is a large cavity occupying most of the upper lobe. Its walls are compressed and smooth. In one portion of it there is an opening 1 cm. in diameter leading into the pleural cavity. Elsewhere in the lung there are small caseous foci and small groups of miliary tubercles.

Left lung is slightly adherent to the pleura. The lung is voluminous, congested in the apex, some thickening and contraction of the pleura and there is present a small cavity 2 cm. in diameter with caseous, partly calcified contents.

The heart is much displaced to the left side. The apex is 4 cm. to the left of the nipple.

The liver is displaced downward, being entirely below the ribs. It is also pushed very much over to the left side. The fissure of the liver is 6 cm. to the left of the median line.

In this case the tuberculosis was of seven months' standing before the development of the pneumothorax. The latter evidently devel-

oped at night a few hours after an evening in which his cough had been severe. The patient died about three months later.

Four days after the onset every sign of pneumothorax was present: the distension of the side, the displacement of the heart, the marked movable dulness, the succussion splash; especially noticeable was the intense amphoric respiration; the metallic tinkling and coin sound were well heard.

At first the liquid exudate was not great in amount, but at autopsy the amount was large. The cavity occupied almost the whole of the upper lobe and the perforation was a large one 1 cm. in diameter. It is interesting that although the right lung was entirely collapsed and functionless, yet the symptoms were never severe even at the onset.

CASE 6.—*Pyopneumothorax of left side due to tuberculosis of the lungs with cavity formation, also to a perforating tuberculous ulcer of the œsophagus; gangrene of the pleura; operation; death; autopsy.*

Annie K., white, 33 yrs. **Med. No. 2260.**

Admitted Aug. 3, 1892.

Transferred to surgical side Aug. 6, 1892.

DIAGNOSIS.—*Pulmonary tuberculosis; pyopneumothorax.*

COMPLAINS of "fever, shortness of breath, cough."

FAMILY HISTORY.—Tuberculosis on father's side.

PAST HISTORY.—Negative. Has always been well.

PRESENT ILLNESS.—Began January, 1892, with "influenza," which was followed by "inflammation of the left lung." She improved, but has had a cough with yellow expectoration since then. About thirteen weeks ago she was taken with a sudden pain in the left side, which increased on taking a deep breath. The cough did not increase. She was in a hospital seven weeks, was aspirated once. Has felt better, but never well. A swelling surrounded the place where the needle was inserted and the place was painful.

The cough has been "dry."

PHYSICAL EXAMINATION.—The patient lies quietly in bed, respirations 24, pulse 92.

The left side of the chest bulges, the intercostal spaces are obliterated. The cardiac pulsation can be seen in the 2nd, 3rd and 4th right intercostal spaces, in the 2nd to within 3 cm. of the mammary line. The cardiac dulness extends from the sternal border to the right mammary line.

The right side on percussion is clear except over the heart. Pulmonary resonance extends to the lower border of the 6th rib in the mammary line.

On the left side the note is full and tympanitic. Flatness below begins at the 6th rib in the recumbent, at the 3rd in the erect position. In the back flatness begins at a point 3 cm. above the angle of the scapula. On

the back opposite the 8th and 9th spaces is a fluctuating area about 7 cm. in diameter which pulsates with respiration.

Auscultation.—The respiration is clear throughout the right side. At the left apex it is enfeebled and passing into the flat area becomes distant with a tubular quality. Over the left front there is nowhere characteristic amphoric respiration, but there is most beautiful metallic tinkling. On shaking the patient one gets a well-marked succussion splashing. The coin sound is very well marked. Vocal fremitus is absent over the flat area. Heart sounds are quite clear.

The liver is not depressed, dulness reaching the costal margin.

The sputum is purulent, foul-smelling and contains tubercle bacilli.

The urine contains a faint trace of albumin.

The exploratory puncture showed purulent fluid in the pleural cavity. The patient was transferred to the surgical side.

While on the medical side the temperature was for three days 101, and then fell to normal.

Surgical No. 1739.

Transferred from the medical side August 6, 1892.

Died August 23, 1892.

DIAGNOSIS.—*Chronic empyema; pyopneumothorax; tuberculosis of the lung.*

The OPERATION was performed August 6. 5 cm. of the left 6th rib was excised between the nipple and the axillary line. The pus did not flow at once, the pleural cavity being full of air. The odor was very offensive. Turning the patient on the side a moderate amount of dirty greyish pus escaped. While coming out of the ether the patient appeared to vomit a small amount of the same material. The wound was drained. The patient recovered well from the immediate effects of the operation.

August 8. The pleural cavity is irrigated daily with normal salt solution. The washings appear to contain coagulated milk and have a strong odor of whiskey.

August 18. The irrigation is continued. Small masses of food appear in the washings from the pleural cavity. The wound gaps widely and on looking in one sees the collapsed lung and the upper surface of the diaphragm; also the beating of the heart. The apex beat is in nearly the same position as before the operation. The patient is weak and emaciated.

August 23. The patient lost strength rapidly and died to-day.

The temperature was irregularly elevated from 99 to 102.7 until August 21, after which time it was subnormal.

Autopsy No. 334.

ANATOMICAL DIAGNOSIS.—*Tuberculosis of lymph glands of neck; tuberculosis of lungs; tuberculous pleurisy with pyopneumothorax; perforating ulcer of œsophagus and gangrene of left pleura. Tuberculous ulcer of œsophagus.*

Body 161 cm. long, much emaciated. Absence of rigor-mortis. In the left thoracic wall is an aperture 4 x 2 cm. in size, skin incision being 4 x 8

cm., a portion of 6th rib being deficient. Operation begins 6.5 cm. below nipple and 1 cm. to right of nipple line. It extends obliquely upward and backward and terminates at a point 9.5 cm. below axilla.

In the left pleural cavity there is 500 cc. of milky, foul-smelling fluid in which float flakes and coagula, mostly white in color. Thin yellowish pus adheres to posterior wall of cavity and there are small collections in three or four other places.

Left lung is compressed and much reduced in volume. The costal pleura as well as the pleura covering the lung is dark gangrenous in appearance, the surface is rough, of a mottled grey color, with numerous hæmorrhagic spots. The lower part of the lung is not so dark in appearance as the upper part. Over the entire upper lobe the pleura is thickened, œdematous, very dark in color, the ground substance being brownish black. Everywhere over this part of the surface it is beset with hæmorrhages. This surface on scraping can be partly removed until the underlying ground substance can be seen and this has a similar dark color; the pleura is everywhere thickened. The adhesion to the diaphragm is firm and complete. About 3.5 cm. from the posterior border and about midway of the compressed lung is a small opening through which a probe can be passed into the lung substance. On incision this is found to lead into a small cavity, the walls of which are composed of a greyish white, granular, caseous looking material. On section the lung is airless, dense, deeply colored, greyish black, and in the lower lobe are a small number of bronchi, dilated, with hæmorrhagic walls, containing a thick, purulent material.

Right lung is free from adhesions. Pleura delicate in most places. The surface is moderately pigmented and very irregular to the feel. Between the lower and middle lobes, midway between anterior and posterior borders, is a circumscribed, granular deposit, easily removed, the pleura underneath having lost its luster in part, due to a fibrinous deposit. Similar deposits elsewhere over entire lung. On section the consistence is increased, the surface is roughened, mottled dark red and light grey, with areas of deeper congestion over the surface. Besides this there are numerous circumscribed areas of consolidation elevated above the surface, greyish white with intervening deep red lines. In the apex is a small nodular cavity with thickened caseous walls communicating directly with a bronchus. These consolidations and circumscribed caseous areas are found everywhere throughout the lung.

Œsophagus.—The œsophagus for a distance of 4.5 cm. lies exposed in the upper part of the pleural cavity. At one point, which is 2.5 cm. above the aorta at the point where the left carotid artery branches off, is a circumscribed erosion 4 x 2 mm. leading directly into the œsophagus. The edges of this ulcer are smooth, the tissues all around it being œdematous and hyperæmic. At other places, in this exposed part of the œsophagus. small and superficial erosions occur, not penetrating the muscular coat. The œsophagus is covered with a granulation tissue. The described ulcer

communicates with the left pleural cavity. On section the œsophagus shows besides the perforating ulcer, another ulcer in the mucous membrane and passing into the muscle, about the same size as the former but not perforating. The edges of this ulcer are also smooth and contain no caseous material. Maggots in ulcer.

This patient gave a history of phthisis of eight months' duration. Thirteen weeks ago a sudden pain was felt in the left side. She was in a neighboring hospital for seven weeks. During this time she was aspirated once. Following the aspiration the point of puncture was swollen and painful. She was admitted to this hospital with the definite signs of pyopneumothorax. The left side bulged, the heart was entirely to the right of the sternum. On percussion this side was hyperresonant and there was extreme movable dulness of three interspaces. In the back is a fluctuating area, hence the case may represent one of pyopneumothorax necessitatis. No typical amphoric respiration was at any time present. The metallic tinkling was beautiful. The succussion splash and the coin sound were well obtained. The patient was transferred for operation. When the pleural cavity was opened only an offensive gas escaped. The pus was evacuated when the patient was turned on the side. Immediately after the operation the patient vomited material resembling the contents of the pleural cavity and ever afterwards on irrigating the chest food was found in the pleura, showing a communication between this cavity and the œsophagus. Such was the condition found at the autopsy. The perforation in the lung was $3\frac{1}{2}$ cm. from and about midway in the posterior border of the compressed lung and led into a small tuberculous cavity. The left pleura was gangrenous. A perforating ulcer of the œsophagus was found.

CASE 7.—*Pyopneumothorax of the left side, due to phthisis with cavity formation; death; autopsy.*

(This case was reported by Dr. Hewetson in the *Johns Hopkins Hospital Bulletin*, Vol. III, p. 120.)

John J., colored, 50 yrs. old. Waiter. **Med. No. 2368.**

Admitted Sept. 8, 1892.

Died Sept. 29, 1892.

Clinical Diagnosis.—*Phthisis Pulmonalis; Pneumothorax; Aneurism of the Aorta.*

Complained of "great weakness, shortness of breath, and cough."

Family History.—His mother and three brothers died of phthisis. Another brother now has this disease.

Past History.—Negative. He had diseases of childhood, and malaria many years ago.

Present Illness began with a cough about twelve months ago. This has been continuous, he has lost weight and strength; expectoration abundant, nummular and often blood-tinged. He has chilly feelings sometimes and fever every night for one week, but seldom sweats. He has done no work for nine or ten weeks. His alcoholic and venereal histories are bad.

Physical Examination.—Sputum. Contains numerous tubercle bacilli. It is purulent, greenish-yellow and streaked with blood.

Urine.—Gives a distinct Diazo test. There is a faint trace of albumin.

Respiration shallow, 20 to the minute, pulse 116, temperature 101.4.

Thorax.—The left side is fuller and expands less than the right on quiet respiration. The right clavicle is particularly prominent.

In the 1st right interspace is the pulsation of an aortic aneurism (the further signs of this condition will not be here given).

Sept. 27. On percussion the note over the left front and axilla is hyperresonant. On the right side it is of much higher pitch with a somewhat tympanitic quality at the apex and as far down as the third rib. The cracked pot sound is obtained just below the clavicle on the right side. Behind on this side from apex to base the note is higher pitched, especially so at the apex. On the left side behind, the note is fair from apex to base, being higher pitched in the interscapular region. A slight movable dulness is made out at the lower left axilla.

Auscultation.—At the right apex breath-sounds are exaggerated in intensity, expiration is prolonged, having a cavernous quality just below the clavicle, while inspiration is accompanied, as far down as the third rib by a shower of fine crackling râles. On the left side at the apex respiration is much feebler, few râles being heard. Below the clavicle breathing is still feeble but inspiration is accompanied by distant metallic râles. On forced inspiration the quality is amphoric. This is most marked on coughing when the metallic quality is pronounced. This amphoric quality of deep breathing is heard over the whole front and axilla as far down as the eighth rib in the anterior axillary line. Voice sounds have an amphoric ring on the left side. Tactile fremitus is increased over the right side, and is still felt over the left side.

Backs.—On the right side at the apex the breath sounds are exaggerated with râles on coughing. At the base the râles are more numerous. On the left side at the apex breathing is somewhat distant, accompanied by wheezing râles. In the interscapular region it is decidedly tubular in character and inspiration is accompanied by fine, crackling râles. This is true to the base along the vertebral column. External to the scapular line, respiration is amphoric and the râles metallic.

Heart.—There is no cardiac dulness to the left of the sternum. The relative dulness extends from the right sternal border to within 2 cm. of the right nipple. The sounds are loudest in the 2nd space in the right nipple line.

The Hippocratic succussion splash not obtained. The coin sound is well heard over the left front and axilla but not in the back. There is slight movable dulness at the left base.

The patient died on Sept. 29, 1892.

Temperature during his stay was quite irregular ranging from 96° to 104°.

The urine contains a trace of albumin. The Diazo test is given.

Autopsy No. 351.

Anatomical Diagnosis.—*Aneurism of aorta. Tuberculous pyopneumothorax. Chronic tuberculosis of lungs with cavities. Caseous pneumonia. Chronic passive congestion. Chronic interstitial orchitis.*

Body 158 cm. long, emaciated.

Right lung bound down by moderately firm adhesions. Both layers of pleuræ of the left lung are much thickened. In the left pleural cavity there are about 50 cc. of thick, yellowish, flocculent pus, and air. Lungs are somewhat compressed but not greatly. There was an escape of gas on opening the thorax.

Left lung.—Layers of pleura are much thickened, bound down firmly behind and to the diaphragm. On anterior surface about the middle of lung, somewhat nearer to anterior than to posterior border, is a circular opening about 2 mm. in diameter, communicating with the pleura and with a cavity in the interior below. Other cavities occur throughout the substance of the lower lobe. There are also cavities in the upper lobe and general peribronchial conglomerate tuberculosis. In addition to this around the caseous masses in upper lobe, the lung substance is more solid and gelatinous in appearance and there are areas of caseous pneumonia, some measuring 5.5 x 1.5 cm. The cavities in the lower lobe have a distinct capsule, sometimes quite thick and lined with granulation tissue, red and covered with caseous masses. In dependent end of lower lobe are quite large cavities and the lung tissue throughout is solid and represents smooth, grey, pigmented consolidation. The pleura covering the lower lobe is covered with thick fibrinous exudate, easily removed, leaving a congested surface beneath. The pleura contains distinct miliary tubercles.

Right lung presents a cavity at apex measuring 5 cm. in diameter, communicating with smaller cavities lined with granulation tissue, similar to those in left lung. The apex especially and the upper part of upper lobe are traversed by bands of fibrous tissue. Throughout the upper lobe are masses of peribronchial caseous tubercles, but no other cavities. In the other lobes are masses of peribronchial tubercles with no cavities. Lungs are injected.

Heart.—A dissection was made in situ before opening the thorax, exposing the 1st interspace close to the sternum, where the pulsation was seen. The pleura costalis is thickened and adherent to the pleura pulmonis. The margin of the lung is exactly 3.5 cm. from the edge of sternum and the intercostal artery and vein pass over the site of the pulsation. In the sternal half of this space is exposed, after the removal of artery vein and connective tissue, an artery. On removing the 1st rib and clavicle the exposed vessel forms a prominent aneurismal-like sac 4.5

x 3.5 cm., the longest diameter being transverse. The pericardium comes above the border of the lower margin of the 1st rib and on opening it there is below the 1st interspace the appendix of the right auricle, which extends over a distance of fully 7 cm. from the sternal margin. The mass proves to be an aneurism of the arch of the aorta, projecting just below the origin of the innominate. It looks to be about the size of a small orange and has a firmer nodular projection just below the 1st rib.

The great interest in this case lay in the diagnosis of the aneurism, but for the discussion of this point the reader is referred to Dr. Hewetson's paper.

The diagnosis of left-sided pyopneumothorax due to phthisis with cavity formation was easily made on admission from the distension and relative immobility of that side, the full percussion note, the absence of cardiac dulness, the distant amphoric respiratory sounds, the metallic tinkling and the coin sound. The phthisis was of at least one year's duration, but when the pneumothorax developed one cannot say.

At autopsy air rushed from the left pleural cavity. The perforation was about the middle of the anterior surface of the lung and communicated with a cavity.

The very small amount of fluid may well explain the absence of any succussion splash.

CASE 8.—*Left-sided pyopneumothorax, entire; due to phthisis with extensive cavity formation; death; autopsy.*

John B., white, 33 yrs. **Med. No. 3690.**

Admitted Feb. 8, 1894.

Died March 28, 1894.

DIAGNOSIS.—*Pulmonary tuberculosis. Intestinal tuberculosis.*

COMPLAINED of "cough and severe cold."

FAMILY HISTORY.—Negative.

PAST HISTORY.—Negative.

PRESENT ILLNESS.—This began eighteen months ago with profuse hæmorrhage from the mouth. The blood was of a bright red color. He felt well until three months ago when a cough began, which has since then become very severe, especially at night. He has had chills at irregular intervals. He has dyspnœa on slight exertion, pain in the left side on taking a deep breath, and profuse purulent expectoration. No hæmoptysis, no night sweats, no digestive disturbances. Is emaciated.

Urine.—Diazo test marked. There was no albumin.

The sputum was laden with tubercle bacilli but no elastic tissue was found.

Physical Examination.—The patient is somewhat pale and emaciated. The thorax is a fair example of Trichterbrust. Pulse 112 to the minute, respiration 28. There is deficient motion of the left lower half. On percussion there is good resonance on the right side. On the left upper front there is well marked dulness especially over the 3rd rib with cracked pot resonance in the 1st and 2nd i. s. Vocal fremitus is increased on the left side.

Throughout the whole left front respiration shows a markedly tubular modification with abundant fine medium and coarse moist râles. In the 1st and 2nd spaces breathing is more tubular in character with sharper finer more resonant râles, and with whispering broncophony. Throughout the right front breathing is slightly modified and accompanied by fine moist râles at the end of inspiration. The right back is clear except at the upper part where are heard a few fine moist râles on inspiration. The respiratory murmur everywhere throughout the left back is enfeebled and of a tubular character accompanied by fine moist râles.

Heart.—Not dislocated.

Liver.—Not palpable.

March 5 (Dr. Osler). The patient has not been so well. Fever of an irregular type marked periods of low temperature. No change in the physical signs.

March 27 (Dr. Thayer). The patient died at 4.45 a. m.; for several weeks the patient has been in a desperate condition—apparently moribund—and no examinations have been made. He has had morphia freely. At the autopsy a pneumothorax was found to have developed since the last note.

The temperature during this illness was very irregular, ranging from 96.8 to 103 or over.

The respiration ranged from 30 to 40 per minute, but during the last four days was constantly above 40.

Autopsy No. 506.

Anatomical Diagnosis.—*Tuberculosis of lung with cavity formation; chronic pleurisy. Perforation of left lung; pneumothorax. Intestinal tuberculosis. Tuberculosis of mesenteric glands and bronchial glands and liver. Acute spleen tumor. Chronic diffuse nephritis.*

Body 165 cm. long, somewhat emaciated. There is slight œdema of ankles. Surface is pale. Rigor-mortis in upper and lower extremities. Pupils wide and equal.

Diaphragm on the right side at 4th space, on left side at 7th rib.

On puncturing the left pleura beneath water there is a free escape of gas. The left pleura is a large air space occupying the lower and superior border zones of the pleural cavity. The lung is compressed and in the floor of the cavity are 600 cc. of yellow, tolerably dense serum in which float white flakes. Both layers of pleura are much thickened and over entire anterior surface of the upper lobe they are adherent to each other. The lower lobe of lung is a mere appendage and is firmly attached to the

upper lobe and in both lungs are masses of caseation, apparently regularly distributed over the lungs. In the upper lobe is a cavity embracing the apex, its long diameter being 6 cm., while transversely it is 5 cm. The walls are quite smooth excepting the soft caseous areas which adhere to it. Along the side are elevated ridges which correspond to bronchi. The bronchi communicate freely with the cavity, which over anterior and superior portions is covered by hardly more than double layer of thickened pleura. Over the remainder of the lung are many bronchiectatic cavities. The pleura over the diaphragm is much thickened and covered with flakes of adherent yellow fibrin. At the base is an opening in pleura almost circular, 8 mm. in diameter, which communicates with two small openings on inner surface with the lung.

Right lung is voluminous, pleura is thickened, especially over the upper lobe, but much less than over the left lung. The lung is bound by old adhesions to thorax wall over upper lobe and in the pleura are numerous minute ecchymotic points. On section the upper lobe is the seat of disseminated conglomerate tuberculosis, the tuberculous masses being arranged about the bronchi. There are also small but distinct bronchiectatic cavities in the upper lobe and upper part of lower lobe. The middle lobe is likewise the seat of peribronchial tuberculosis, but the lower lobe over one-half its extent is free. The lung tissue here is œdematous and congested and this is likewise the case between the tuberculous masses. In bronchi is sticky muco-pus.

Clinically this case presents little of interest, since no physical examinations were made after the onset of the pneumothorax. The tuberculosis was of about eighteen months' duration and the left lung was nearly destroyed. The perforation was at the base, the pneumothorax entire, the pus small in amount.

CASE 9.—*Pyopneumothorax acuta of the left side, due to chronic tuberculosis with cavity formation; death; autopsy.*

Lewis H. L., 41, white. **Med. No. 3955.**

Admitted June 4, 1894.

Died June 28, 1894.

Clinical Diagnosis.—*Pulmonary Tuberculosis. Pneumothorax.*

Complained of cough and progressive loss of weight.

Family History.—Good.

Past History.—He has lived an intemperate life as bartender. His venereal history is also bad.

In 1879 he says he had an attack of "malaria;" a six weeks illness in bed with numerous chills and night sweats, and loss of 30 pounds weight. For the past two years he would catch cold easily, but otherwise has felt well.

Present Illness.—Ten months ago he began to cough a good deal, mostly at night and early in the morning, with scanty expectoration,

white, sometimes yellow. After three or four months he began to lose weight, and now weighs 52 pounds less than ten months ago. Never night sweats or hæmoptysis or fever. He has been getting progressively weaker since the onset. During the past six months the sputum has been getting more abundant, purulent, never blood tinged.

Urine.—No Diazo test.

The sputum is mucopurulent and contains very many tubercle bacilli and elastic fibers.

Physical Examination.—The patient is emaciated and sallow, respirations from 36 to 40 per minute, pulse 104.

Thorax well shaped. There is deficient expansion on the right side. The percussion note is high pitched at the right apex, dull over the right back. It is clear on the left side.

Auscultation.—The breath sounds are clear at the left apex in front, but from the 5th rib extending into the axillary region are heard numerous medium-sized râles with inspiration and expiration. At the right apex the breath sounds are cavernous, particularly at the 2nd i. s. Expiration is prolonged; there are medium-sized râles on quiet breathing, on coughing large gurgling râles. In the mammary region the breath sounds are high pitched with some piping râles. Numerous moist râles in the right axillary region. Medium-sized râles throughout the upper part of the right back; the breath sounds are high pitched and whiffing, not tubular. Moist râles at the left apex behind.

Heart and abdomen negative.

June 28, 1894. At 8 p. m. last night the patient seemed to be in about usual condition. At 2 a. m. he was found sitting up in bed, livid, extremities cold, respirations shallow, 40 to the minute, and pulse almost imperceptible. He complained of distress and fulness in the epigastrium, not more on one side than on the other. He said he had been very short of breath for about half an hour. Under free administration of morphia he rallied somewhat, extremities became warm, and the pulse of better volume, but at 4.30 he died suddenly. During the whole time he referred all the distress to the epigastrium.

The temperature during his stay in the hospital, at first 100 to 103°, was irregular and gradually dropped, ranging during the last two days from 97 to 100.

The urine was negative. The Diazo test was not present; no albumin.

Autopsy No. 537.

Anatomical Diagnosis.—*Pulmonary tuberculosis with cavity formation and caseous pneumonia; acute serofibrinous pleurisy; pyopneumothorax; old pleural adhesions and thickenings; patent foramen ovale; splenic enlargement; puckerings of liver; tuberculous ulcerations of intestines (including rectum); fistula in ano; old hæmatocele(?); chronic interstitial orchitis and periorchitis; chronic passive congestion; syphilis(?); old leg fracture with deformity; cholelithiasis; cirrhosis of liver.*

Body 173 cm. long, emaciated, rigor-mortis well marked, livor-mortis

in dependent parts. Pupils are even. Right leg in middle third markedly deformed, owing to old fracture. The upper fragment points downward and outward, the lower upward and outward, leading to shortening and angular deformity.

Diaphragm on right side at 4th interspace, on left side at 7th space, bulging down into the abdominal cavity.

On opening left chest gas escapes violently. The left pleural cavity is distended with air and frothy fluid, the latter amounting to one-fourth the volume of this side of the thoracic cavity. The left lung is retracted toward its root and is much shrunken. It is hindered from more complete retraction by old adhesions at apex and to diaphragm. The fluid, besides being frothy, is turbid and contains a few greyish-white particles in suspension. The costal pleura on this side is everywhere thickened, also along the posterior border of the lung and at apex, and along the fissure between the two lobes it is also thickened. Both parietal and costal pleuræ are covered with a fresh adherent fibrinous exudate, which varies in thickness, being most marked over lower lobe and anterior margin of upper lobe. The fibrinous exudate is also abundant between the lobes on this side and along the lower anterior part, where they are bound together by recent adhesions. The upper part of the fissure is partly obliterated by old fibrous adhesions. Near apex of lower lobe behind is a perforation of the pleura measuring 6 x 3 mm. and communicating with a small cavity into the lung and from which the curdy sanguineous contents of the cavity freely pass through into the pleural sac. Scattered over the pleura of both upper and lower lobes are greyish-white patches of necrosis. On cutting into these they are found to correspond to nodules of caseous pneumonia or to small cavities in the lung substance. The surface of the lung where the exudate is thin, can be seen to be marked off everywhere by interlobular markings of coal pigmentation. Nodules of solidification can also be felt from the outside throughout the substance of both lobes. On section at apex is a cavity, the size of a pullet's egg, with purulent, caseous contents and irregular, roughened, worm-eaten walls. Throughout the rest of upper lobe are multiple nodules and minute cavities varying in size and appearance, some of them are typical nodules of broncho-tuberculous-pneumonia, with caseous center and gelatinous periphery, and thus resemble nodules in bronchopneumonia in stage of red and grey hepatization. The little cavities appear to have resulted from the breaking down of the areas of consolidation, many areas being seen with only a little softening in the center. These cavities are evidently of very recent formation. Their contents is greenish-yellow and purulent; sometimes in addition they have minute curdy particles. The lower lobe of this lung contains numerous nodules, similar to those described in the upper lobe, but softening is not so frequent. Pulmonary arteries are normal. Bronchi are hyperæmic and contain mucopurulent sputum.

Right pleural cavity is dry but there is a fresh fibrinous exudate over

all lobes and between the lobes. The lung is voluminous. The upper lobe is almost completely solid and on section shows multiple nodules of consolidation and minute cavities, the intervening lung substance being loosely solidified by fresh gelatinous pneumonia. The middle and lower lobes are hyperæmic and in addition show a few patches of bronchopneumonia.

In this case the perforation was a large one, 6 x 3 mm., situated at the apex of the lower lobe, behind, of the left lung and communicating with a small cavity. The pyopneumothorax was almost entire, and the frothy pus occupied about one-fourth of the pleural cavity. When the pleura was opened the gas "escaped violently," hence at the time of autopsy at least the cavity did not open into an open bronchus. His tuberculosis was of very long standing, yet the more acute process began about ten months ago.

The onset of pneumothorax was sudden one night with collapse, dyspnœa and epigastric pain. He died fifteen hours later.

CASE 10.—*Pneumothorax acuta of the left side, due to phthisis with extensive cavity formation; death; autopsy.*

James A., aged 41, white. **Med. No. 3963.**

Admitted June 7, 1894.

Died June 11, 1894.

Clinical Diagnosis.—*Pulmonary tuberculosis; pneumothorax; carcinoma of stomach.*

The patient complains of pain in the abdomen and cough.

Family History.—In the father's family there were several deaths from tuberculosis.

Past History.—The patient has never been seriously ill. His habits have been good.

Present Illness.—This dates from January 1, 1894, though the patient had a cough for some time before this. Since January the cough has been very severe accompanied by sharp pain in the left chest and much mucopurulent bloody expectoration. The patient has had two or three hæmorrhages but does not remember the dates. Since February he has suffered from diarrhœa, the stools being mucous and bloody with much pain and tenesmus. The appetite has been good. There has been a gradual loss of flesh but there have been no chills. He has had night sweats and occasional headaches.

The urine on admission contained a very faint trace of albumin, there was no Diazo reaction. The sputum contained numerous tubercle bacilli.

Physical Examination.—The patient is propped up in bed, emaciated, not very anæmic. The respirations are quite abdominal. The pulse is soft, regular in force and rhythm. Thorax is flat, expanded in the lower portion. Expansion is slight but apparently equal. Both clavicles are

markedly prominent. The percussion note is markedly impaired at both apices above and below the clavicle. Below this on both sides is hyperresonance. The breath sounds at both apices are harsh with prolonged expiration. At the right apex inspiration is accompanied by numerous fine moist râles. Vocal fremitus is increased. There is marked whispering bronchophony at the right apex. Below at both bases is dulness. The breath sounds in the interscapular region have a tubular modification, otherwise they are the same as on the front. The apex beat is not visible but the sounds are best heard in the 5th space just inside the nipple. They are clear. Hepatic flatness extends from the 6th rib to the costal margin. The abdomen is negative.

June 11, 1894. This morning the patient is lying on his right side; there is marked dyspnœa. The pulse is 160. The left side is rather more prominent than the right. Resonance is everywhere full and tympanitic covering the entire cardiac area on the right side. As before noted there is well marked dulness at the apex. At the extreme left apex respiration is feeble and almost absent. In the 2nd left space and along the border of the sternum can be heard the faintest amphoric sound. Below this it is almost absent. In the back there is the same full tympanitic resonance, while the respiration is everywhere extremely feeble. The coin sound is not well marked. The heart sounds are best heard at the right of the sternum in the 4th space. The patient is rapidly getting worse. The shortness of breath is increasing. The pulse is feeble. He died in about three hours after the preceding note.

The temperature on admission was normal, but on June 10 rose to 101.3.

Autopsy No. 531.

Anatomical Diagnosis.—*Pulmonary tuberculosis with cavity formation; tuberculous pleurisy; acute pneumothorax; tuberculosis of larynx and trachea; ulceration of tonsil; chronic tonsillitis; ulceration (tuberculous) of large and small intestines; disseminated tubercles in liver and kidneys; tuberculosis of mesenteric lymph glands; primary medullary cancer of lesser curvature of stomach; metastatic cancer in liver and lymph glands. Arterio-sclerosis.*

Body 160 cm. long, emaciated, rigor-mortis well marked, livor-mortis in dependent parts. Grœnish discoloration of abdomen and thoracic walls.

Diaphragm on right side 4th space, left side 5th rib.

Left lung collapses partially. Right lung firmly bound down by old adhesions. There are old adhesions at apex of left.

Left lung is free from adhesions excepting at apex. The upper lobe is diffusely invaded with miliary tubercles, more numerous in upper and anterior part than in lower part of lobe. There are old adhesions between upper and lower lobes. The upper and posterior portion of lower lobe is partially solidified and filled with tubercles. On section the upper lobe shows a number of minute areas of bronchial and peribronchial tuberculosis. Between the tubercles the alveoli partly contain air and are partly filled with exudate. The organ is red in color. In the upper part of the

lobe are many small, recent excavations. Some of them communicate with one another, the surface is covered with tuberculous granulation tissue and some show minute strands of tissue running across them. Contents of the cavities is yellowish and purulent. One of them has reached the surface of the lung in front, 4cm. from apex. There is an area measuring 15 x 15 cm. where the lung substance has been completely eroded, covered only by the thin visceral pleura. About the center of this is a perforation of the pleura 3mm. in diameter. The upper part of the lower lobe is almost solid with tubercles. These are evidently of bronchial distribution. Many of them are grey and some of them have yellow centers. Scattered tubercles occur in the lower part of this lobe with here and there islands of bronchial tuberculosis. Bronchi are hyperæmic. Pulmonary vessels show an occasional yellow patch in intima.

Right lung is firmly bound down by old adhesions between two layers of pleuræ and between the lobes. In front at base is a firm mass measuring 8.5 cm. in length, which consists of an encapsulated cheesy exudate, whose walls are made up of firm fibrous tissue, in places one-half of a cm. in thickness, cartilaginous in feel. The upper lobe, middle lobe, and upper part of lower lobe are consolidated and filled with miliary tubercles. At the apex is a multilocular cavity the size of a walnut which communicates with a bronchus. There are partitions in the cavity, which is divided into two large and many small cavities. The walls of the cavities are comparatively smooth and the contents consists of cheesy material and frothy fluid.

This case of acute pneumothorax of the left side developed in the course of phthisis with extensive cavity formation of at least six months' duration. He died in about three hours after the onset. Clinically the physical signs were: increasing dyspnœa; distension of the left side; marked displacement of the heart; a full tympanitic note on percussion over the entire side; but the respiratory sounds were very feeble notwithstanding the fact that the perforation was found open at autopsy and the coin sound could not be obtained.

CASE 11.—*Pyopneumothorax, total, on the right side, due to pulmonary tuberculosis with multiple ulcerated and bronchiectatic cavities; death; autopsy.*

John A., colored, 38 years old. **Med. No. 5490.**

Admitted Oct. 21, 1895.

Died Nov. 1, 1895.

DIAGNOSIS.—*Pulmonary Tuberculosis. Pneumothorax.*

COMPLAINS of "cough and shortness of breath."

FAMILY HISTORY.—Negative.

PAST HISTORY.—Negative. He has always had good health as a sailor. His habits have been fairly good.

Present Illness.—The onset was gradual. In May of this year he began to have a slight cough. At first the expectoration was scanty, mucopurulent and tenacious, but within two weeks of onset became quite profuse. No fever was noticed at first. For the past four weeks he has had slight sweats, especially at night. No dyspnœa was noticed until the last three weeks. This the patient said appeared quite suddenly. Cough has persisted since the onset and now is very troublesome, especially at night. No hæmoptysis. For the past three weeks the patient has had pains in the epigastrium, especially after meals. He vomits often after a severe paroxysm of cough. No œdema. Micturition is of abnormal frequency. Appetite has been lost.

The sputum is of considerable amount, slightly blood-stained, and contains a great many tubercle bacilli.

Physical Examination.—Patient is emaciated. Pulse 180. The chest is flat. Expansion is deficient over the entire left front. The percussion note is dull over the entire left side, being almost flat in the anterior axillary region. On the right side it is of a distinct full tympanitic quality. Tactile fremitus is enfeebled over the entire right side, and is not increased over the left side.

Auscultation.—Over the entire left front and back the respiratory sounds have a tubular quality, expiration being prolonged and distinctly tubular. The sounds are rather distant except in the supraclavicular and upper mammary regions. At the end of inspiration and during expiration numerous fine crackling and fine mucous râles are heard, also loud piping râles. The voice sounds are only slightly increased over the left front and the whispered voice is nowhere distinctly transmitted.

Right side.—Over the entire right side front and back the breath sounds are very feeble. During inspiration fine musical piping râles are heard and at the end of inspiration very fine crackling râles. In the right axilla expiration is slightly tubular and has a peculiar metallic quality, but is not truly amphoric. Whispered voice sounds are nowhere transmitted.

Heart.—The impulse is plainly visible in the 5th and 6th i. s., about 2 cm. outside the nipple line.

The coin sound is intense.

Nov. 1, 1895. The patient during the past forty-eight hours has been growing rapidly weaker, coughed much, suffered great pain. He died to-day.

The temperature was at first 104° and gradually fell to normal on the day of his death.

The urine contained a trace of albumin. The Diazo was negative.

Autopsy No. 734.

Anatomical Diagnosis.—*Phthisis pulmonalis; ulcerative and bronchiectatic cavities with perforation of right lung and right-sided pyopneumothorax. Peribronchial tuberculosis and tuberculous pneumonia (diplococci in exudate). Tuberculosis of larynx and œsophagus. Tuberculosis of intestines. Acute splenic tumor. Parenchymatous degeneration of viscera.*

Body 170 cm. long, strongly built, moderately well nourished. Rigor-mortis in both extremities. No subcutaneous œdema. Abdomen moderately distended. Chest, particularly on right side, prominent.

The right pleural cavity opened under water. By shifting the body an escape of gas takes place through the water. On removing the intercostal muscles fluid is seen to fill the right pleural cavity to within 3 or 4 cm. of the ribs and sternum. The space remaining evidently represents the air area. The lung is compressed and occupies the posterior and median portion of the cavity. The mediastinum is displaced to the left; a semicircular line passing from the first rib to the junction of the xyphoid cartilage with the gladiolus, with its deepest part to the left of the median line, above at the junction of the ribs and left border of the sternum, corresponds with the mediastinum. The right pleural cavity contains fully 2000 cc. of fluid, which above is clear and yellow, but below is thick and purulent; curds are mixed with the purulent fluid. The left lung is free from adhesions. The pleura not especially thickened. Tubercles can be felt beneath the membrane.

Right lung.—The upper portion of the right lung is firmly bound to the chest wall, the middle and lower lobes are free. Both layers of pleuræ are thickened. The greatest thickening corresponds with the adhesions over the upper lobe. The parietal pleura is thicker than the remainder of the visceral pleura. Over the middle and lower lobes the thickening merely gives an opacity to the pleura; elsewhere it is dense and fibrous looking. Pus and fibrin are scattered over the entire free surface of the pleura and tubercles are visible beneath this layer. The lung is greatly compressed. It is firm and airless. In the middle lobe, nearer the posterior than the anterior border, where it joins the upper lobe, there is a circular perforation of the pleura, measuring 1 cm. in diameter. The edges of the perforation are necrotic and covered with a thick yellowish-white exudate. This perforation leads into a cavity and this into a medium-sized bronchus. The upper lobe of the lung on section is riddled with cavities. No very large ones occur. These are merely small ones the size of a hazel nut, or an almond. Some of these are distinctly bronchiectatic; others appear to be ulcerated cavities. The intervening lung tissue is very dense, much pigmented, airless. The middle lobe contains several cavities much resembling those in the upper lobe. It is also compressed. The lower lobe contains fewer cavities. This lobe is shriveled but not so dense in consistence as the upper lobe. Tubercles, for the most part of a conglomerate nature, are seen between the cavities. The bronchi contain pus. The vessels at the base of the lung are free.

Left lung is free from adhesions. The pleura is not especially thickened. The lung is voluminous. Anterior edge emphysematous; also apex. On section a very few small (hazel-nut) cavities are found in the upper lobe. The apical part of the lung is emphysematous. The lower part of this lobe and the lower lobe is in part consolidated. The consolidation is dark red in color, perfectly smooth and moist. The tubercles are for the most

part conglomerate in character and often caseous. Central bronchial perforations are seen in the middle of the consolidated portion and about it. Bronchi contain pus. Vessels at the base of the lung are free.

Displacement of the heart is not marked.

Liver is pushed down on the right side. It extends 10 cm. below costal margin.

In this case the pneumothorax developed during phthisis of about five months' duration. The onset was sudden but not severe, dyspnœa and epigastric pain being its chief features. The physical signs were not marked; although the fistula was large and perhaps open the amphoric respiration was not present. Death followed about four weeks from the onset of the pneumothorax. The right lung was airless; the pyopneumothorax total; the perforation was situated at the superior border of the middle right lobe and was 1 cm. in diameter. The lung was riddled with cavities, many of which were bronchiectatic.

CASE 12.—*Pyopneumothorax due to phthisis of the right lung; death; autopsy.*

Thomas J., colored, aged 20. **Med. No. 13,768.**

Admitted December 2, 1901.

Discharged December 28, 1901.

DIAGNOSIS.—*Pleurisy with effusion, tuberculosis, thickened pleura.*

The patient complained of shortness of breath and pain over the heart.

FAMILY HISTORY is negative.

PAST HISTORY.—Has always been a strong, healthy, hard-working man until the present illness. He has had no cough. His habits have been good. His work requires heavy lifting and in a heated atmosphere near the smelting furnaces.

PRESENT ILLNESS began last spring. Patient caught what he called a "heavy cold," which confined him to bed for about a month. This attack came on suddenly with high fever, sharp pain and aching all over. There was severe cough. During this attack he spat up a little blood on one or two occasions. For the last couple of months he has noticed gradually increasing intermittent pain in the lower thorax, particularly on the left side, and this has been accompanied by increasing dyspnœa. The pain is uninfluenced by respiration but is made worse by coughing. Two weeks ago last Saturday his pain, cough and dyspnœa became so severe that he was obliged to stop work and has done nothing since. For the preceding week before this he had been especially exposed in working. There have been no chills. He has had night sweats every other night. He has lost 20 pounds. The expectoration has never been abundant.

PHYSICAL EXAMINATION.—The patient is well nourished. The thorax is well developed. The right side is fuller than the left. The intercostal

spaces show no difference. Expansion is markedly less on the right side, which practically does not move at all. Percussion note is everywhere flat throughout the right side, except over the 5th interspace in front and at the extreme base behind where there is some resonance. The resonance is well marked over the left side except in an area in front over the lower sternum in which there is tympany. The breath sounds are well heard throughout the left side, are everywhere harsh. The right side presents a marked contrast, as scarcely anything is heard except over the area mentioned behind in which the breath sounds are heard. They are slightly nasal. Definite coin sound cannot be obtained. Succussion not obtained.

The heart's impulse is in the 5th interspace 12 cm. from the mid-sternal line. The slight area of cardiac dulness is outside of the above mentioned area of tympany, but over the base of the heart the note is tympanitic. The vocal fremitus is absent on the right side. The liver is not palpable. The sputum shows no tubercle bacilli.

December 3. The patient was tapped and 1300 cc. of sero-purulent fluid removed. After the tapping the tympanitic area over the front is replaced by lung resonance. There is still resonance at the base. No movable dulness made out and no succussion.

December 5. The patient was again tapped and over two litres of fluid drawn out.

December 6. There is a great amount of immobility. The back is flat all the way up, the fremitus is marked at the base. Feeble distant breathing is still present. There is a slight friction rub at the extreme base. The left side is 1½ inches less than the right side. There is movable dulness in front of the right chest.

December 14. The patient's right chest seems filling up again. There is definite impairment of the note just beneath the clavicle to the 3rd rib. Below this there is definite movable dulness. The patient was discharged December 28 much improved. While in the hospital his temperature was fairly normal; urine negative.

Second admission September 18, 1902. **Med. No. 14,834.**

Died September 23, 1902.

DIAGNOSIS.—*Pulmonary tuberculosis; pyopneumothorax.*

The patient COMPLAINED of pain in the left side and cough.

Present illness began four weeks ago while the patient was at work with a sudden hæmoptysis of bright red frothy blood. The patient kept at work that day, though he felt weak. He said he felt the hæmorrhage from the left lung. The next day he did not work, but two days later was back in the fields at work again. The cough has become much worse. The sputum has been blood-tinged but never any fresh hæmoptysis since onset. Weakness has increased. Since September 13 he has been dizzy and unable to stand any length of time. He went to bed and pain developed in the left side of the chest. Has been in bed ever since. He has not felt feverish, but has had a sweat every night. The cough has become increasingly severe, especially in the day time and when he lies on the left

side. The appetite is good; bowels regular. He has been losing weight very rapidly in the last month.

PHYSICAL EXAMINATION.—The patient looks very ill; the tongue is coated; mucous membranes are of fair color.

Chest.—Expansion is markedly less on the right side. Over the upper front and axilla there is a quality of dull tympany. Otherwise percussion note is resonant throughout. The percussion note is clear over the right side. There is marked difference between the two sides on auscultation. The breath sounds are distant over the right front and rather suppressed in the right axilla. Over the left front and axilla the breath sounds are loud and harsh. Expiration is prolonged and there are numerous coarse râles, principally with inspiration. Practically the same condition is found in the upper backs. In the lower right back are a few fine râles on inspiration. Vocal fremitus is somewhat diminished on the right side. The heart sounds are loud and booming; pulse 100. The point of maximum impulse is in the 5th space, 9½ cm. from the midsternal line. Abdomen is natural. Spleen is not palpable. The sputum is not abundant, is mucopurulent, nummular; tubercle bacilli are very abundant, no elastic tissue.

September 22. The patient is in a worse condition to-day. His respirations are very labored. Coarse râles are heard everywhere in both phases of respiration.

September 23. At one o'clock this morning patient was breathing with some difficulty. Pulse was weak, 128 to the minute, very small volume. The patient is almost comatose, is roused with difficulty. In spite of heavy stimulation coma developed and the patient died at 3.30 a. m. The sputum was slightly more abundant yesterday, but contained no blood. The urine contained a trace of albumin. No Diazo. Temperature irregularly elevated between normal and 104.8.

Note after death. The pyopneumothorax was not recognized during life.

Autopsy No. 2001.

ANATOMICAL DIAGNOSIS.—*Pulmonary tuberculosis; perforation and collapse of right lung; right-sided pyopneumothorax; cavity formation, left upper lobe. Diffuse conglomerate tuberculosis of left lung. Thickened pleuræ, right and left. Tubercles in liver and kidneys. Cloudy swelling of myocardium. Fatty liver and kidneys. Multiple macular-like scars of skin (due to burns).*

The body is that of a moderately well-nourished colored man, 173 cm. long. Pupils are equal. Rigor-mortis well marked. Scattered over the body and over both extremities, back and front, are a large number of small scars, white in color with a dark periphery, and varying in size from pin-head to half a pea. There is no definite scar on penis. Diaphragm reaches the fifth rib on both sides.

On opening the thorax the left pleural cavity is found to contain about 100 cc. of turbid fluid. There are old fibrous adhesions between the apex of the left lung and parietal pleura. The right lung is collapsed and is firmly adherent to the mediastinal structures. The right pleural cavity

contains about 300 cc. of thick greenish pus, occupying a large thick-walled opening extending from the diaphragm to apex of pleura.

Left Lung.—The pleural covering is everywhere thickened, especially over the lower lobe, where it is about 0.5 mm. thick and is rough. The color of the lung is greenish grey. The consistence of the upper lobe is firm, non-crepitant except at the border, and contains scattered nodules of consolidation throughout. The lower lobe is everywhere crepitant, but less so than normal. No nodules of consolidation are felt in the lower lobe. On section the cut surface of the upper lobe shows a mottled dirty green and red appearance. There is a cavity filled with thick, dirty, necrotic material, irregular in shape and about the size of a hen's egg in the lower part. Scattered everywhere through the parenchyma are conglomerate and discrete tubercles. Many of the former are caseous. The cut section of the lower lobe is light red, moist, and shows everywhere scattered through it discrete and conglomerate tubercles. No congestion is met with in the lobe. The mucous membrane of the bronchi is red, congested and covered with muco-pus.

Right lung is collapsed. The pleural covering is everywhere thickened, in most places being 1 to 2 mm. thick and is light in color. The consistence of the upper lobe is non-crepitant. The middle and lower lobes are slightly crepitant. No areas of consolidation are met with in either lobe. On section the upper lobe is dark in color and a piece sinks in water. No tubercles are seen in this lobe. The cut surface of the middle and lower lobes are lighter pink in color. No tubercles are met with except near the base of the lower lobe. On the postero-lateral aspect of the lower lobe of the lung there is a depressed area in the pleura about the size of a silver dollar. From the center of this area a sinus leads down which on being followed is found to branch in several directions but no opening into a branch was found. The bronchi of right lung are similar to those in left lung. Apparently the sinus comes from a small cavity in the lower lobe.

In this case the pneumothorax was discovered only at autopsy. The total duration of the phthisis was roughly seventeen months. On the first admission pneumothorax was suspected but not diagnosed. At the second admission nine months later the signs on the right side were, limited motility, distant breath sounds and diminished fremitus. At the autopsy the right lung was collapsed, almost airless, and but 300 cc. of pus were present. The fistula was found and was of the valvular variety.

If, as is possible, pneumothorax was present on the second admission, then this case is one of those described by Laennec in which the hyperresonant note misleads the clinician.

CASE 13.—*Pneumothorax of the left side, pure, closed, due to caseous pneumonia; death; autopsy.*

Angelina N., colored, 20 years old. **Med. No. 9836.**

Transferred from the gynecological side April 28, 1899.

Died May 12. 1899.

Diagnosis.—*Pneumothorax; caseous tuberculous peribronchial pneumonia.*

Complains of pain in the lower abdomen and the small of the back.

Family history shows several cases of tuberculosis.

Past History.—She has never been a strong child. Has had measles, whooping-cough and rheumatism (fever with swollen and painful ankles and wrists). She had malaria when 12 years old for four weeks; diagnosis made by a physician. Chorea in 1894. She never had scarlet fever, typhoid fever, pneumonia, mumps, chicken-pox or diphtheria. She has always had a sick headache off and on since a child.

Following her confinement two years ago she has had a dull aching pain in the abdomen, especially about the navel.

Her venereal and alcoholic history are of the worst.

Has always worked very hard.

Present Illness.—The patient traces the onset to October, 1898. At that time, following a venereal sore, she had a generalized skin eruption associated with a swollen, painful ankle, wrist and elbow, which confined her to her bed for ten days. Night sweats appeared at this time and have been almost constant since. They are not associated with chills and fever. The patient has been growing weaker since the onset. She has had pain in the bones but no swelling of the joints since the onset. The patient has been unable to work since October. About four weeks ago, following a shaking chill, she had a sharp cutting pain in the left side near the nipple, which was increased by deep respiration and coughing. She was more comfortable when lying on her right side. If on her left side she had coughing and pain. Has not had much expectoration, although she has had a cough since March, 1899. She never spat up any blood. She has had pain in the abdomen localized in the umbilical region for the past four weeks, a dull ache increased by coughing and moving about. She has had fever for the past two weeks in the afternoon and in the evening, but no chills since the onset. No herpes; no cardiac distress; no œdema of the feet; epistaxis twice last week; nausea and vomiting occasionally after coughing. The appetite has been poor. She has lost weight and strength since the onset.

Physical Examination.—The patient is fairly well nourished; no dyspnœa; no cyanosis; coughs frequently; not anæmic.

Thorax is well formed and symmetrical. The clavicles not especially prominent. Expansion slight but equal.

Left Side.—Vocal fremitus well felt; percussion note a trifle impaired in the right supraclavicular region. Below this in the first interspace the note is high pitched and of a cracked pot character. Auscultation.—The sounds in the supraclavicular fossa are harsh blowing in character; expiration is prolonged. Below this the sounds become more distinctly am-

phoric and the voice sounds are rather nasal. Numerous fine moist râles are heard in the front. In the lower left axilla a friction rub is heard with numerous fine moist râles.

Right Side.—Vocal fremitus is well felt. The note on percussion is impaired. Auscultation.—Fine moist râles heard at the apex and lower axilla. In the apex the breath sounds are slightly harsh with prolonged expiration.

Backs.—Vocal fremitus is well felt. The percussion note distinctly impaired in the right subscapular region. The breath sounds are enfeebled over this region. Fine moist râles at both bases.

Heart.—Apex beat is present in the 5th space 7½ cm. from the midsternal line. Cardiac dulness and sounds are normal. Abdomen is natural. No œdema of the limbs. There is general glandular enlargement.

May 1. Marked impaired resonance over the left front with marked amphoric respiration in the first interspace. At times a sound suggesting a cracked pot note is elicited. There is pure amphoric breathing in the first, second and third spaces. Crackling râles at the height of inspiration with whispering pectoriloquy and bronchophony. The abdomen is not distended.

May 3. The percussion note is still impaired over the upper left front, its amphoric quality is not so marked as formerly. The vocal fremitus is diminished. The breath sounds are enfeebled and impaired and have lost their characteristic tubular amphoric quality. Expiration still has tubular modification. The marked exaggeration of spoken and whispered voice-sounds previously heard has largely disappeared. The percussion note is everywhere impaired in the left axilla and back. The breath sounds are markedly suppressed. A few very fine crepitant râles are heard at the angle of the left scapula.

May 4. The respiration is short and halting. There is an expiratory grunt. The left side looks fuller, particularly in the lower zone of the chest. The left shoulder is higher than the right. The intercostal spaces are not seen on either side. Expansion is distinctly less on the left side. There is no visible cardiac impulse to the left of the sternum. The pulsation to the right of the sternum is seen in the second and third fourth and fifth interspaces extending just beyond the parasternal line; also pulsation in the epigastrium. The resonance over the right chest is of natural pitch. It is rather hyperresonant in the upper left front above the fourth rib. In the axilla it is resonant and of higher pitch and not so full in volume. The resonance to the left of the chest obliterates the heart dulness and extends as far as the right sternal margin. The respiratory murmur on the right side is somewhat exaggerated. The breath sounds on the left are almost entirely suppressed; what is heard has a slight amphoric quality particularly marked in the upper front. The voice sounds on the right are natural; on the left are much weakened and of slight amphoric quality. No adventitious sounds. There is general suppression of the breath sounds throughout the back. General hyper-

resonance throughout the left back. General diminution of tactile fremitus on the left side. The heart's dulness extends 7¼ cm. to the right on the fourth rib and extends upward to the second interspace. The sounds are not distinctly heard until the left sternal border is reached. They are heard most distinctly to the right of the sternum, increasing in intensity from below upward. The second sound is loud at the base and reduplicated. The coin sound is well heard in the left front and back. At the region of the apex a pleuro-pericardial friction rub is well heard.

May 6. This morning at two o'clock the patient had a severe shaking chill.

May 10. The chest was aspirated with an apparatus to collect air over water; 500 cc. of air were removed. Immediately after aspiration the relative cardiac dulness extends from 5 cm. on the right to 5 cm. to the left in the fourth interspace. The visible impulse to the right has diminished. The sounds are heard to the left. There is no definite localization of maximum intensity; well-marked pericardial as well as pleuro-pericardial friction rub is heard. To the left of the sternum the note is quite amphoric in the left front. Voice sounds and respiratory murmur markedly amphoric in the back; the percussion note is of a higher pitch and of Skodaic quality.

Pressure in the chest before aspirating + 4 mm. Hg. Deep inspiration caused a drop to — 8 mm. Hg. On coughing the oscillations are very marked. Air was easily aspirated, coming in gushes synchronous with expiration and coughing.

Pressure at the end of aspirating, — 7 mm. Hg.

Gas removed; 503 cc. gas (at 20° C). $CO_2 = 5.65$ per cent.

May 12. This morning the patient seems brighter than she has during the last week. The dyspnœa and respiratory distress are much less marked. Pulse 96 to the minute and fairly strong (2.30 P. M.). This evening at 7.15 the patient became rather short of breath and seemed in distress. A needle was inserted in the eighth interspace of the axilla, considerable air escaping with a whistling sound. She died quietly at 7.35 P. M. The temperature while the patient was in the hospital was at first regular and elevated, between 102.5 and 104.5. It much resembled that of typhoid fever. It remained fairly regular, but gradually dropped somewhat.

The sputum was examined seven times for tubercle bacilli, but none were found.

The urine contained a trace of albumin, with hyaline and granular casts.

Autopsy No. 1361.

Anatomical Diagnosis.—*Pneumothorax. Pulmonary tuberculosis of abdominal lymphatic glands. Tuberculosis of left lung. Caseous peribronchitis. Caseous pneumonia. Chronic diffuse nephritis, slight. Parenchymatous degeneration of kidneys. Pelvic adhesions. Small cyst in ovary.*

Body of a slenderly built young woman, 161 cm. in length. Rigor-mortis well marked. Panniculus adiposus fairly well preserved. Over thighs and shins there are numerous depressed pigmented scars.

Diaphragm on right side reaches 4th space. Unfortunately not measused on left side until after intercostal muscles were punctured. Puncture was made under water and a rather profuse escape of gas occurred. The gas had a somewhat foul odor. On opening the thorax the left lung was found much contracted and the pleural cavity contained a blood-stained fluid which probably entered on the escape of the bubbles of air. Pleural surface somewhat roughened. Pleura on right side smooth and glistening. No excess of fluid.

Right Lung.—Pleural surface smooth. On section the lung is uniform throughout the middle and lower lobes, rather moist and dark red in color. Everywhere crepitant. In the center of the upper lobe there is a nodule 1.5 cm. in diameter, which is firmly consolidated, rather pale in color, slightly granular and surrounded by a hæmorrhagic zone and somewhat raised above the surrounding surface. Vessels and bronchi clear.

Left Lung.—There is an extensive but irregular consolidation. An attempt was made to discover the mode of exit of the air into the pleura by forcing air into the bronchus, the lung being submerged in water, but there was apparently no opening. The surface of the lung shows a slight roughening and deposit of a thin layer of fibrin. At the lower posterior portion of the upper lobe and scattered throughout the lower lobe there are especially firmly consolidated areas which are pretty sharply outlined and have a greyish color. The whole lung is quite firmly consolidated with the exception of the anterior edge of the upper lobe and several irregular areas behind in the lower lobe which can be insufflated. On section the areas described above as sharply outlined are seen to correspond with masses reaching down into the substance of the lung which are also sharply circumscribed and are centrally softened and spongy, grey and apparently necrotic. The bronchi throughout are dilated, more especially toward the peripheral portions of the lung and are much thickened, the thickened walls having an opaque yellowish-white appearance. The intervening lung tissue is firm and somewhat moist. It is greyish and translucent and quite airless. There are minute greyish yellow specks scattered through it and a slight roughness suggests the possibility of the presence of tubercles. There are no evidences of cavity formation. The vessels are clear of thrombi as far as can be seen. Bronchi not especially congested. They contain a greenish pus which is loaded with tubercle bacilli. The infarct-like areas described above are not large. They measure from 5 mm. to 3 cm. in diameter.

This interesting case of pure pneumothorax supervened on a case of caseous pneumonia without cavity formation. The tuberculosis was of at least seven months' duration. The onset of the pneumothorax was May 3 or early May 4, with almost no subjective symptoms, but marked changes in the physical signs: the percussion note became hyperresonant; the heart moved far to the right; the coin sound was obtained. But in this case without an open fistula

(it must have early closed or at least remained valvular, no trace of it was found at autopsy) the breath sounds were much suppressed and heard with an amphoric quality, and no metallic phenomena were present. The writer was able to clearly demonstrate the presence of air in the chest, to measure the pressure and collect the gas for analysis. At the autopsy the case contained practically only fetid air.

CASE 14.—*Left-sided pyopneumothorax due to phthisis of a little over one month duration; death; autopsy.*

Joseph M., aged 20; colored. **Med. No. 14,950.**

Admitted October 19, 1902.

Died October 31, 1902.

Clinical Diagnosis.—*Pulmonary tuberculosis; tuberculous pneumothorax; tuberculous peritonitis.*

Complains of cough and shortness of breath.

The family history is negative.

Past History.—He was healthy as a child and has had no diseases until the present illness. There has been no cough, no dyspnœa, no hæmoptysis. He has worked hard for the past six or eight years. Habits are good.

Present Illness.—This began in July, 1902, after exposure. The patient woke up in the morning with hoarseness and cough. There was no pain in the side at onset, no hæmoptysis. Up to this time he says he was perfectly well. There was at first no dyspnœa. The cough grew worse and the patient expectorated considerably. He had fever and sweats at night. Late in August, 1902, he began to get short of breath. The dyspnœa increased gradually up to admission. There was sudden onset. Its development was associated with no pain in the side or other symptoms that would attract his attention to one side more than to the other. Throughout the present illness the patient has been able to keep in a recumbent posture. Throughout September and until three days before admission the patient was able to work. He thinks he has had fever and has often had night sweats during the past few weeks. About two weeks ago he first noticed shreds of blood in the sputum and this small amount has been present occasionally since. There has been no hæmorrhage. The cough has been especially frequent lately. The sputum has not increased much. There has been no pain, but gradually increasing weakness. On the day before admission he first noticed swelling of the ankles.

Physical Examination.—Oct. 20. The patient is lying on the left side. There is some dyspnœa. The thorax is rather rounded. The intercostal spaces are more marked on the right side. The expansion is less on the left. Visible cardiac impulse is all to the right of the sternum. The note is hyperresonant on percussion over the right side. Over the left it has less resonance than normal, but is not tympanitic, nor is it markedly dull. It varies considerably, being quite tympanitic in the 3rd, 4th and 5th inter-

spaces inside the left mammillary line. Outside it is more impaired. The right back is hyperresonant. There is some impairment everywhere over the left back. The vocal fremitus is diminished throughout the left side; is present on the right. The breath sounds are everywhere harsh but clear throughout the right side. Everywhere over the left side the breath sounds are rather feeble. Inspiration is slightly high-pitched but expiration has a most marked bell-like amphoric quality. There is well-marked coin sound and a well-marked amphoric succussion both on coughing and on shaking the patient.

Heart.—Point of maximum impulse is apparently in the 4th right interspace 8 cm. from the midsternal line. The heart sounds are much alike, and are fairly regular. The abdomen is markedly distended.

Red blood corpuscles, 3,900,000; leucocytes, 9000; hæmoglobin, 55 per cent.

October 31. The patient has been gradually becoming weaker and there has been a little nocturnal delirium. He lies persistently on the left side. There is incontinence of urine. There has been no especial change in his condition. At five o'clock this evening he seemed as usual. The pulse is 120, of fair quality. The cough has been slight and there was practically no dyspnœa. The patient was apparently rational. At 5.25 P. M. he was found dead. There were no signs of hæmorrhage, no change in position. Up to this morning there had been no change in the physical signs. In the purulent fluid found in the pneumothorax cavity post mortem there were an extraordinary number of tubercle bacilli. The urine contained a trace of albumin with hyaline and granular casts. The temperature has been constantly elevated for the first five days, ranging from 100 to 103; afterwards from normal to 102.5. The respirations have been as a rule about 40 to the minute.

Autopsy No. 2025.

Anatomical Diagnosis.—*Chronic pulmonary phthisis with cavity formation; perforation of the left pleura; pyopneumothorax; pneumonoconiosis. General miliary tuberculosis; tubercles in liver, spleen, and intestines. Tuberculous peritonitis. Tuberculous laryngitis. Chronic passive congestion of liver and kidneys. Acute splenic tumor.*

Body of a fairly well developed colored man. Scrofulous scar on right side of neck. Marked obliteration of intercostal space on left side with bulging of right side.

The skin is separated from the thorax and is filled with water on the left side. A probe being inserted into the fifth interspace, a large amount of air emanates from the pleural cavity. Anterior mediastinal glands are adherent to the sternum. The left pleural cavity is very large and contains about 500 cc. of thick yellowish pus.

Left lung is firmly bound down to the posterior costal surface and cannot be separated without tearing the lung substance. On removal a cavity is found at the side of adhesions the size of a large hen's egg. Connection of this cavity with a bronchus easily demonstrated with a probe. The lung

is very small, measuring 10 x 15 x 8 cm. The surface is covered with thick, rough pleura and pus. On section the lung substance throughout is hard, black, and pieces from all portions sink in water. Its substance seems to be firm and fibrous.

The pleura of the right lung is bound by a few old adhesions. Lung is rather firm and contains pale areas. The surface is greyish.

The anterior edge of the liver is displaced downward. Depth on right side is at the level of 6th rib, on the left side at the upper border of the 7th.

In this case of left-sided pyopneumothorax the phthisis was evidently of just about one month's duration before the onset of the pneumothorax, the pneumothorax of about two months' duration before death. The lung was found firm and airless. The fistula was not demonstrated at autopsy.

CASE 15.—*Pneumothorax of the right side, due to phthisis; death; autopsy.*

Wm. W., 36 years old, white. **Med. No. 15,259.**

Admitted January 23, 1903.

Died February 10, 1903.

COMPLAINS of pleurisy and a heavy cough.

FAMILY HISTORY.—Mother died of consumption. Otherwise family history negative.

PAST HISTORY.—The patient had the ordinary diseases of childhood. Following scarlet fever at ten years of age he had some swelling of the feet. He had malaria last summer. For the past eight years he has been coming to this dispensary for "nervous prostration." He has suffered from headaches of such intensity that he has had to go to bed. He has had a hacking cough for 12 or 14 years. Expectorates a little white phlegm during the day but nothing at night. Five or six years ago he wrenched his side throwing a baseball and was in bed four or five weeks with a pain in the side. Five years ago had pleurisy in the left side which lasted two or three months. There was a stabbing pain on every motion and on deep inspiration. For the past eight years the patient has had paroxysmal attacks of dyspnœa lasting about a week and coming on every month or so. He knows of nothing that seems to bring on these attacks. During the attacks he gasps for breath. Quite frequently he has had to run to a drug store to beg for something to keep him from dying. He has had palpitation of the heart and pain in the region of the heart ever since he has suffered from "nervous prostration." The patient now suffers from indigestion. His habits have not been good.

The patient is an oyster shucker and has to work in all kinds of weather.

PRESENT ILLNESS.—This began September, 1902. The patient was cracking ice with bare feet and got overheated. Before that time he had not been feeling very well and had a hacking cough. In about a week after this exposure developed a sharp stabbing pain in the right side. He also had

what he called a "rattling in the side." The following morning he tried to work, but was too weak and stopped in about a half hour. Since then he has not worked, but has not had to go to bed. He gradually lost weight, losing fourteen pounds in the first two weeks. During this time he had very little appetite. Five weeks ago he was toasting some bread over a fire when he suddenly felt a smothering sensation in the chest. He had to sit down for want of breath. There was no pain. He did not lose consciousness. In a few minutes he coughed up a large solid lump about the size of the end of his thumb which he says was white like phlegm. Since then his dyspnœa has gradually increased and his condition grown worse. He has no pain in the chest, but suffers much from dyspnœa. He has a cough and expectorates considerable sputum. There is no pain in the heart but some palpitation. The appetite is poor. He has a feeling of fullness and soreness in the stomach.

Physical Examination.—The patient looks ill. There is slight puffiness about the eyes. The mucous membranes are pale. The respirations are 32 to the minute. The thorax is very rounded, rather symmetrical. The interspaces on the right side are fuller than on the left. Expansion is greater on the left. The percussion note over the left front and axilla seems practically normal excepting close to the sternum. Over the right front the note is high-pitched. Below the third rib and extending down to the costal margin and throughout the axilla the note is hyperresonant with an amphoric percussion tone. This also extends to the left of the sternum in the 6th interspace 6 cm. from the midsternal line. Both lungs are hyperresonant. The resonance extends in the back to the 11th rib. The breath sounds over the right side are everywhere distant, a little harsh and not definitely amphoric. Over the left side the breath sounds are rather harsh and high-pitched. There is a very well marked coin sound on the right side and most marked intense metallic succussion. There is practically no dulness in the lowest part of the chest.

Heart.—Point of maximum impulse is in the 6th interspace 15 cm. from the mid-line. There is a marked visible impulse in the 4th, 5th and 6th interspaces outside the nipple. The relative cardiac dulness begins in the 3rd space. Both sounds are heard. The first is followed by a systolic murmur, not especially loud, but rather intense, heard faintly in the axilla. The second sound seems everywhere clear. The pulse is 108. The abdomen is slightly full, rather more so on the left side. On palpation a doughy resistance is present, especially in the upper region. There are no nodules. Elastic tissue and tubercle bacilli are both present in the sputum.

January 26. Dr. Osler: The patient is cyanosed. There is marked dyspnœa, slight pulsation in the veins of the arm. The pulse is good. The point of maximum impulse is far out in the axilla. The heart sounds are well heard over the right side. The breath sounds are distant. In front of the right of the sternum there is quite a loud breezy inspiration. In the posterior axilla scarcely anything is heard. Amphoric breath sounds

and metallic tinkling in an exquisite way are heard after the patient coughs. The fremitus is absent. The coin sound is present in a remarkable degree and can be heard by those standing near the patient. The pericardial friction rub is heard over the heart.

The circumference of the left side is 32½ cm. on the right side, 37 cm. at the angle of Louis. At the juncture of the 4th rib and sternum the circumference on the left side is 34.3, on the right 40½. At the zyphoid cartilage the circumference of the left side is 42 cm., of the right is 38.

January 26. A small aspirating needle connected with a simple water manometer was inserted in the 8th interspace in the mid-axilla. At the end of the first inspiration the intra-thoracic pressure was + 6 cm. of water; at the end of the first expiration + 11 cm. of water. On coughing large amounts of air escaped, so that finally at the end of expiration the pressure was — 2 cm. of water at the end of inspiration and + 2 at the end of expiration, but the pressure gradually returned to a positive one during both inspiration and expiration. On closing the tube and asking the patient to cough the pressures were slightly lowered, but returned gradually to their former level. The pulse during the operation became low in tension and very markedly dicrotic. The dyspnœa was remarkably relieved. The percussion note on the right side after the paracentesis was tympanitic, higher pitched than before, but the limits of the pneumothorax cavity were apparently unchanged. The right border of the relative cardiac dulness is still 5 cm. to the left of the midsternal line, the patient's cyanosis has disappeared and the dyspnœa has considerably lessened, though there is no change in the physical signs. Cough is infrequent.

January 30. Red blood cells, 4,400,000; leucocytes, 14,000; hæmoglobin, 60 per cent.

February 9. Dr. Osler: There are no amphoric sounds nor metallic tinkling.

February 10. For the past week the patient has been growing weaker, with considerable dyspnœa and cyanosis. Yesterday again the Béclère apparatus was used, some gas removed. A few drops of pus also escaped. The patient became weaker and died this evening.

Autopsy No. 2072.

Anatomical Diagnosis.—*Pulmonary tuberculosis with cavity formation; perforation of right lung. Tuberculous pyopneumothorax. Tuberculous pericarditis. Bronchopneumonia, left lung. Bronchitis and tracheitis. Tubercles in spleen. Tuberculous ulcers of intestine. Tubercles in capsule of liver. Intestinal tape-worm. Moderate arterio-sclerosis. Calcification of mitral and aortic valves.*

The body is that of a much emaciated man, 158 cm. in length. Rigor-mortis is present. Pupils are equal. There is slight post-mortem lividity of the dependent parts. In the 8th interspace of right axilla is a small incision into which a probe can be passed about 1 or 2 cm. In the 5th and 6th interspaces of the right anterior chest wall are old puncture marks. There is no scar on penis.

Diaphragm 6th rib on right side, 6th rib on left side.

The right pleural cavity contains about 2200 cc. of a dirty, greenish-yellow, foul-smelling fluid. The right lung is retracted and collapsed. The parietal pleura is covered with fibrino-purulent material, which leaves a rough granular surface. The left lung is bound to the diaphragm by firm fibrinous adhesions. It is also adherent to the pericardium by adhesions which can be readily broken up. The right side of the pericardium forms part of the cavity of the right pleura. The pericardial cavity is obliterated, the parietal and visceral layers of the pericardium being adherent; the adhesions can be easily broken through.

Left lung is voluminous and has a mottled appearance, grey areas alternating with dark livid areas. The upper lobe is roughened from old adhesions and has many sub-pleural ecchymoses. The lung is in general crepitant, with larger and smaller areas which seem airless. Bronchi contain a considerable amount of muco-purulent material. Pulmonary artery is clear. On section the lung has in general a rather greyish appearance, with many irregular areas standing out above the general level of the section. These areas are purplish-yellow in color, dry, granular, often surrounded by a rim of injection. No areas of distinct caseation are seen in this lung, nor are there any definite tubercles to be made out. On section, however, a group of small, yellow tubercles is met with, which are firm and project above the cut section.

Right lung is bound by one or two adhesions to the upper part of the chest, but lung as well as pleura is lined by thick, necrotic, yellow material in which there is granulation tissue. Lung is uniformly airless. Along the upper lobe the surface is granular and there are many fibrinous bands which bind it to the chest wall and here the lung is also bound to the mediastinal tissue. At this point there is a small opening through which a probe can be passed into the cavity. At the root of the lung another cavity at the posterior side of the apex is broken into. The bronchi are very much enlarged and contain a considerable amount of bloody purulent material. In the apex there is a large cavity and a probe enters the lung substance in almost any direction. On section the lung is filled with conglomerate tubercles of various size, many of them showing caseation. The cavity in the anterior upper border is about 3 cm. in diameter, irregular in outline and contains a lot of dirty, reddish-grey material.

The liver extends 8 cm. below the tip of the ensiform cartilage in the mid-line and 11 cm. below the costal margin in the right mammillary line.

The points of interest in this case are: the extreme distension and dyspnœa, yet with but slight intrathoracic pressure, not nearly what would be expected. The metallic tinkling was beautiful and could be heard for some little distance below the costal margin.

Circumscribed Tuberculosis Pneumothorax.

CASE 16.—*Pyopneumothorax necessitatis, circumscribed; perforation; death.*

John W. J., colored, aged 23 yrs.; sailor. **Med. No. 1966.**
Admitted March 23, 1892.
Died April 2, 1892.

Clinical Diagnosis.—*Pyopneumothorax; pulmonary tuberculosis.*

Complains of "cough, hoarseness, shortness of breath."

Family History.—Good; no tuberculosis.

Past History.—A temperate man; denies venereal disease. Has always been strong and healthy. His habits have been good.

Present Illness.—Eighteen months ago he was shot in the right side by a 32-caliber revolver; the bullet is said to have remained in the chest. The external wound healed in two weeks, but the patient has "never felt well inside" since. He has had a cough ever since the injury, and expectoration, which for the first week was bloody, then purulent until about five months ago, when for two weeks it was again bloody. The sputum seems to come more fully when lying on the left side. Since the last hæmoptysis he has had fever and dyspnœa, and during the past three months night sweats. Appetite poor; no nausea or vomiting; bowels regular. Micturition increased in frequency. He has lost in weight.

He has been hoarse during the past seven days. Yesterday for the first time he noticed his feet were swollen.

Seven days ago he noticed a swelling in the lower part of the right side of the chest. Five days ago a needle was inserted and the patient states a small amount of blood was withdrawn.

Physical Examination.—The patient is propped up in bed; is pale and anæmic and emaciated. Respiration, 40; pulse, 140, dicrotic, of low tension. Thorax: The two sides seem equal, except at the lower right front, where is localized bulging. There is somewhat more motion on the left side. In the fifth and sixth interspace, just under and inside the right nipple, is an area where with each inspiration the chest wall sinks and with each expiration is blown out, as if it were a piece of parchment. On palpation at this point there seems to be a deficiency of the sixth rib, and air can be felt apparently just below the skin (emphysema); during inspiration at times can be felt a curious rattling as if of fluid. On this prominence are two scars, one recent and the other pigmented.

On percussion the right side above is perhaps slightly more tympanitic than the left. At the fifth rib is relative dulness which extends over above-mentioned area; over the lower right axilla the dulness is marked.

Auscultation.—The respiration is harsh over the left front, a number of fine râles heard at the end of inspiration. Over the right front the murmur is exaggerated, expiration much prolonged and accompanied by wheezing râles. Over the pulsating area there is a curious amphoric quality of respiration, inspiration being accompanied by metallic râles and expiration by loud sonorous râles, prolonged and apparently near the ear. In the lower axilla the breath sounds are feeble, distant and amphoric; the voice sounds are not increased. If the patient is sitting up during some of the violent fits of coughing a peculiar splashing sound is heard in the region of the pulsating area.

Backs.—The upper right back is possibly a little more tympanitic than the left. Expiration is more prolonged. Below the angle of the scapula there is marked dulness and diminished fremitus. The respiratory sounds are distant but of pure amphoric quality. The coin sound was not obtained. The left back is clear on percussion; the respiratory sounds are very harsh, accompanied everywhere by fine medium and coarse bubbling râles.

Heart.—Point of maximum impulse is felt in the fifth interspace just inside the nipple line. The pulmonary resonance extends over the whole cardiac area. The first sound at the apex is replaced by a soft, systolic murmur, propagated a short distance towards the axilla. The second pulmonic is accentuated.

Further History.—In a few days the above-mentioned pulsating tumor burst, discharging with each respiration considerable pus. The pus since then has escaped in smaller quantities, but with each expiration air is forced through the opening with considerable power.

April 2. The patient has rapidly failed and this afternoon died.

The temperature chart for the ten days the patient was in the hospital shows an irregular fever ranging from normal to 104.6°.

The urine contained a faint trace of albumin.

Autopsy No. 290.

Anatomical Diagnosis.—*Circumscribed pyopneumothorax following gunshot wound. Tuberculous cavities in lungs. Tuberculous bronchopneumonia and caseous pneumonia. Induration of the lungs. Tuberculosis and anthracosis of the bronchial glands. Old pleuritis and pericarditis. Obliteration of the pericardial sac. Heart hypertrophy. Tuberculous ulceration of the intestine. Simple catarrhal ulcers of the large intestine. Tuberculosis of the mesenteric and colic lymph glands. Tuberculosis of the spleen. Fibroma in the kidney. Fatty degeneration and swelling of epithelium of kidney and liver. Miliary tuberculosis of liver. Chronic miliary tuberculosis.*

Body 160 cm. long, sparsely built and well nourished. Rigor-mortis well developed in the lower extremities; not so much developed in the upper extremities. Over the buttocks is a bed sore, 10 x 3 cm. Over the left thigh there are a few cicatrices, movable over the subcutaneous tissue. On the right side in mammillary line, 5 cm. below the nipple, there is an aperture 1 cm. in transverse diameter. The underlying tissues are infiltrated and sero-pus was emitted from the cavity when the drain was removed. It communicated with the pleural cavity.

Left lung bound down by adhesions, especially at its posterior portion and at base. Right lung adherent to the diaphragm and behind. Pericardium adherent to pleuræ.

Right lung is voluminous, has a slaty grey color and a dense irregular feel. On its anterior surface and extending to its base is a circumscribed abscess, limited by parietal pleura in front, the visceral pleura behind, and the diaphragm below. The size of this cavity, which is globular in form,

is 10 x 8 cm. It extends, however, below under the diaphragm for a distance of several cms. The contents of this cavity consists of serous blood-stained pus, containing coagula and air. The cavity communicates with the external air by means of the opening into the pleural cavity, 5 cm. below the nipple. On section the lung presents a very slaty and more or less mottled appearance, given by numerous areas of opaque, yellowish-white masses of caseation. In the apex of the lung, which is indurated, there is a small cavity 1 to 2 cm. in diameter, about which are large conglomerate tubercles, which are also around the bronchi. Caseous pus can be squeezed from the bronchi. The tissue intervening between the caseous areas and the cavity is redder, with streaks of slaty color. The middle and lower lobes of the lung show extensive areas of peribronchial tuberculosis with consolidation about some of them. There are numerous discrete tubercles, some miliary, others conglomerate. A piece of lung tissue cut out from this portion sinks in water. In other places the consolidation is absent, the lung contains air and floats. A few small cavities are also present in these lobes. The abscess has a distinct capsule formed by a membrane with thick walls. The walls are rough and on the surface are distinct elevations and depressions, some of the latter being 2 to 3 mm. in depth. A red granulation tissue forms the main lining of the cavity.

Left lung is voluminous. In the apex is an irregular worm-eaten cavity with caseous walls, 3 to 5 cm., embedded in the substance of the lung. About these the lung tissue has increased density, and there is an extensive tuberculous bronchopneumonia. The entire upper lobe is the seat of a tuberculous bronchopneumonia and the intervening lung tissue, between the affected bronchi, is of a slaty grey color. The tubercles are miliary and conglomerate. The lower lobe is likewise the seat of tuberculous bronchopneumonia, and caseous pus can be squeezed from the cut surface of the bronchi. There are smaller cavities in the lower lobe and small areas of caseous pneumonia are present. The pleura is thickened by old fibrous tissue, but one patch of fresh fibrinous exudation over the consolidated lung beneath.

Pericardial sac is obliterated.

Margin of liver one finger's breadth below free margin of ribs.

This very unusual case of pyopneumothorax necessitatis deserves careful consideration. It was the result of a bullet wound eighteen months previously, since which the patient "has never felt well inside." Seven days before admission the local empyema began to point and five days before admission a doctor tried to aspirate, as a result of which the air may have entered. However this may have been, the abscess contained both air and fluid on admission, as shown by the subcutaneous emphysema, the succussion splash. The abscess perforated. He died ten days after admission. The lungs were

extensively diseased and with cavities, but this local pyopneumothorax cavity 8 x 10 cm. in size connected with none. The origin of the air is doubtful, but may have been gas producing bacteria.

CASE 17.—*Right-sided encapsulated pyopneumothorax, due to tuberculosis with extensive cavity formation; death; autopsy.*

Sophie R., aged 42, white. **Med. No. 393.**

Admitted February 25, 1890.

Died March 10, 1890.

CLINICAL DIAGNOSIS.—*Phthisis pulmonalis.*

The patient COMPLAINS of cough with expectoration, fever, loss of weight and strength, shortness of breath, and hoarseness.

FAMILY HISTORY is negative. The patient is slightly *non compos mentis* and therefore gives a poor history.

PHYSICAL EXAMINATION.—Pulse, 144; respiration, 55. The patient is anæmic and emaciated. The chest is narrow, the clavicles prominent. There are signs of cavity on the right side from the apex to the 5th rib. Cavernous respiration more intense below than above. There are also marked signs of cavity in the lower and upper axilla. On the left side in front is a small cavity at the apex; below is feeble breathing. Numerous bubbling râles are heard. Below there is dulness to the middle of the scapula on the left side and to the angle of the scapula on the right side. There are signs of cavity in the left infrascapular and scapular regions on the right side from the apex to the base. Very loud gurgling râles are heard over the whole of the right lung posteriorly. Heart dulness extends from the 3rd rib. The sounds are normal. The abdomen is negative. The cough is very distressing. Expectoration is profuse, containing tubercle bacilli and elastic tissue.

March 8. During the past few days the long saphenous vein of the left leg has become thrombosed.

March 10. The patient is much excited, is livid. Pulse 116. The patient died this afternoon at two o'clock. The temperature has ranged from 97.2 to 103.

Autopsy No. 78.

ANATOMICAL DIAGNOSIS.—*Chronic pulmonary tuberculosis with cavities. Pyopneumothorax. Tuberculous ulcers and nodules in small and large intestines. Tuberculous ulcers of trachea. Atrophic patches in kidneys. Laceration and erosion of os uteri externum. Thrombosis of left femoral veins.*

Length of body, 160 cm. Extreme emaciation. Left leg and thigh swollen; measurements around left calf, 27.5 cm.; around right calf, 21 cm.; around middle of left thigh, 35 cm.; around right, 28 cm. Swelling pits on pressure. The femoral and the veins around groin are tense and hard. There are numerous red spots in patches on the skin of

left thigh and leg, which do not disappear on pressure. Rigor-mortis in lower extremities; only in small joints in upper. Double cataract. There are two small ulcers on pinna of left ear; one on the upper margin, the other on inner surface, extending to the cartilage, with very little reaction around them.

Diaphragm on right side reaches the 5th intercostal space; on left side upper margin of 6th rib.

Left pleural cavity free from adhesions except at apex, contains a small amount of clear serum. Right pleural cavity contains, between base of lung and diaphragm, an accumulation of about 425 cc. of turbid, thin, brownish-white, sero-purulent fluid, mixed with flakes of fibrin. This accumulation is encapsulated, the rest of the lung being everywhere adherent. On standing this fluid separates into equal parts, with white pus at bottom and turbid yellow serum at top. Gas also present in this cavity and escapes on opening thorax. No offensive odor.

Left Lung.—There is a little fresh fibrinous exudate on pleura between lobes. Considerable œdema of lung. Upper lobe presents numerous scattered areas, from size of walnut to that of hen's egg, of consolidation, presenting greyish-red granular appearance. Opaque white tuberculous nodules arranged like bunches of grapes. Limitless areas throughout lower lobe, especially in upper part. A dense stellate fibrous caseation at apex. Bronchi contain a good deal of muco-pus mixed with frothy serum; also some scattered tuberculous nodules in air-containing parenchyma.

Right Lung.—The lobes are united by fibrous adhesions. The upper lobe is riddled with cavities, the largest the size of a hen's egg, communicating with bronchi and each other, lined by a thin fibrous capsule coated with caseous material. Between cavities lung substance consolidated by opaque white diffuse and nodular caseous deposits; frequently lobules of lung are separated by fibrous bands and parenchyma composed of breaking-down caseous substance. The lower lobe contains smaller cavities surrounded by caseous masses. Most of lung consolidated with caseous nodules conglomerated and frequently embedded in fresh greyish-red pneumonia. Bronchi contain pus and muco-pus. This lung has very little aerated parenchyma; scattered caseous tubercles appear in the pleura. In the lower part of lateral surface of right lower lobe there is a round hole, 3 mm. in diameter in an extremely thin, delicate, pleural wall of a cavity size of a walnut; edges of opening are smooth. This opening communicates with the encapsulated cavity in the pleura containing serum and pus.

Heart exposed over triangular area 8 cm. at base. Pericardium contains about 50 cc. of clear serum. Lungs adherent over pericardium.

The liver in mid-line reaches to umbilicus; in right mammillary line the lower margin is 10 cm. below ribs.

In this case of chronic tuberculosis the fact that the right lung was riddled with cavities was clinically very evident; the small local-

ized pneumothorax, however, escaped detection, being considered as a cavity. Unfortunately the clinical features are not well described. The perforation at the right lower lobe, the lower lateral surface, was open, communicating with a small subpleural cavity.

CASE 18.—*Pyopneumothorax, circumscribed and very limited at the left base, due to extensive cavity formation. Death. Autopsy.*

George D., 53 years old, white. **Med. No. 2498.**

Admitted October 25, 1892.

Died May 7, 1893.

CLINICAL DIAGNOSIS.—*Pulmonary tuberculosis.*

PAST HISTORY.—The patient has been fairly well until two months ago when the present illness began.

PRESENT ILLNESS began with a cough and gradually increasing pain in the left side which has become quite severe. There has been considerable loss of weight and night sweats. There is no history of hæmoptysis. There has been a great deal of shortness of breath. There are no gastric symptoms.

PHYSICAL EXAMINATION.—The patient is lying on the right side, somewhat emaciated, color good. The lower left chest is prominent, the costal margin protruding. The upper left half of the chest is flatter and moves distinctly less than the right. Respirations are 36 to the minute. Resonance throughout the right front is rather exaggerated. On the left side it is possibly slightly higher in pitch but is clear throughout the front. The breath sounds over the right front are clear but exaggerated, also on the left front. Resonance in the axilla is good but when one reaches the 7th space in the posterior axillary line respiration becomes distinctly tubular with medium crackling râles. Resonance throughout the right back is good. On the left side there is deficient resonance in the lower half, particularly so below the angle of the scapula. Vocal fremitus is present but diminished. At about the middle of the left back respiration is clear and above becomes distant and tubular.

Heart.—Apex beat is in the 5th space just under the nipple. There is no flatness in the cardiac area. The border of the liver is not palpable. The abdomen is negative. The sputum has a heavy sweetish odor. It shows no elastic tissue but many tubercle bacilli. The urine contains a trace of albumin.

December 23. The chest is distinctly flatter on the left side; expansion is decidedly less. Tactile fremitus is well felt but not so markedly as on the right side. On quiet breathing there is everywhere at the left back from the middle of the scapula down and as far as the posterior axillary line tubular breathing. There are no râles. Above, the mid-axilla it loses its tubular character. The cough is loud and echoing. The voice sounds are transmitted with the greatest intensity. On deep inspiration there is a loud, almost amphoric breathing in the left infrascapular region. Above the angle of the scapula there are loud râles heard on coughing. Percussion is clear over the left lung in front.

April 17. The patient has had very little fever for the past month, the temperature rising to 101. He has lost in weight. There is lessened mobility on the left side. The cardiac impulse is seen in the 2nd, 3rd and 4th spaces close to the sternum. The left clavicle stands out prominently. Deep respiration is painful and causes cough. Resonance in the left infraclavicular and mammillary regions. The note is dull. Behind there is dulness from apex to base. Fremitus is not so marked as on the other side. Auscultation: In the left infraclavicular region as low as the 3rd rib are cavernous, almost amphoric breath sounds. The heart sounds are loudly communicated. Below this the breath sounds are distant and tubular with the transmitted heart sounds. In the upper axilla the breath sounds are almost amphoric. In the lower mid-axilla they are very distant. Behind, there is tubular breathing on the left side in the suprascapular and scapular regions, becoming at the angle of the scapula loud and intense and almost amphoric accompanied by splashing râles. Pectoriloquy is marked. There is no coin sound. The dulness has extended remarkably in this case, the change being very striking during the past three weeks.

May 1. The patient is lying on the left side; respirations 40; pulse 112. Throughout the right front and axilla the resonance is distinctly tympanitic. Respiration is harsh and exaggerated. Throughout the left front and axilla there is almost complete flatness. The vocal fremitus is there present but not as marked as on the right. As low as the 4th rib respiration is distant and tubular, below this it is almost absent. In the backs on inspiration there is almost immobility of the left side and nearly complete flatness. Vocal fremitus is everywhere felt, but diminished. The right side is clear. In the left supraspinous fossa is distant amphoric respiration with whispering bronchophony. Below this there is dull tubular respiration until one reaches a point 3 cm. above the spine of the scapula where respiration becomes markedly amphoric and accompanied by loud resonant râles. This amphoric respiration reaches around to the axilla. The coin sound is not heard. The maximum cardiac impulse is in the 4th space at about the parasternal line. The sounds are clear. The hepatic flatness begins at the 7th rib and extends 6 cm. below the costal margin. He died May 7.

The urine shows a faint trace of albumin; no Diazo reaction. The temperature while in the hospital was slightly elevated, ranging from normal to 102 until November 29, when it became practically normal. The slightly irregular temperature fell from normal to 100, continued until April 7, from which date until April 22 it ranged from 98 to 101. It was irregularly elevated after that date.

Autopsy No. 414.

Anatomical Diagnosis.—*Chronic pulmonary tuberculosis with cavity formation; acute caseous pneumonia; chronic tuberculous pleurisy with much thickening. Tuberculous pyopneumothorax. Dilatation and hypertrophy, especially of right side of heart. Arterio-sclerosis. Old perisplenitis with adhesions. Furrowed liver. Acute and chronic nephritis. Tuberculosis of diaphragm with atrophy. Tubercles in kidney. Lipoma of diaphragm.*

Body 154 cm. long. No rigor-mortis. Skin pale. Moderately emaciated.

Left pleural cavity obliterated by adhesions. There are numerous adhesions in right pleural cavity.

Left lung removed with great difficulty. The dense adhesions had to be cut away. Above, corresponding to the 2nd and 3rd ribs and cartilages, there is a fresh pleurisy with slight sero-fibrinous effusion. The diaphragmatic pleura is enormously thickened, measuring in places 6 mm. At the point where the spleen was attached to the muscular coat of the diaphragm, it was wasted and replaced by fibrous tissue. The thickness of diaphragm and pleura is in places 15 mm. The pleura over the postero-lateral aspect of the lung is enormously thickened. Both layers are inseparable. At extreme antero-lateral margin of pleura, there is a small localized empyema, measuring 5 x 5 cm., the walls are rough and ragged. It communicates with the lung by an orifice 3 mm. in diameter which passes directly upward into one of the cavities about the middle of the diaphragmatic surface. This cavity must in reality have been a small pyopneumothorax, as it communicated so freely with the lung. On section, the greater portion of the lower lobe is occupied by a cavity measuring 12 x 9 cm. The content was a very foul-smelling cheesy substance and the material, which, for a few days before his death, he had been too weak to cough up. The walls of the cavity are greyish, in places injected, in others necrotic. It is crossed by numerous trabeculæ. Communicating with it are numerous smaller cavities, occupying the diaphragmatic portion of lower lobe, varying in size from a marble to a chestnut. A very large part of the tissue intervening between these cavities is made up of a firm greyish-white, indurated tissue. The upper lobe of the lung is uniformly airless and on section presents numerous groups of fresh tubercles of all sizes between which the tissue is uniformly infiltrated with a yellowish serum, giving to it a gelatinous appearance. The whole process in upper lobe looks recent (in one or two places the tubercles have fused, forming distinctly caseous masses) and corresponds to the clinical history of an acutely extending infiltration.

Right lung is crepitant throughout. The pleura in places is puckered and over the lower lobe there is a recent pleurisy with fresh lymph and a small amount of sero-fibrinous exudation. On section there are everywhere scattered, particularly about upper and middle lobes, groups of fibroid tubercles, generally in colonies about a pigmented focus of induration. About middle of lower lobe there is an area about 4 x 3 cm. in which tissue is completely consolidated, reddish in color and looks like a fresh pneumonia about groups of tubercles.

In this case which presents several interesting features the pyopneumothorax cavity was a small one, about 5 cm. in diameter, on the left side, communicating by a good sized patent perforation with a much larger cavity of the lower lobe and at about the middle of its diaphragmatic surface. The walls of this pyopneumothorax cav-

ity were the greatly thickened pleura, and it was situated at the right base in the antero-lateral part of the pleural cavity.

This diagnosis was not made during life, it is not strange to say. Almost the entire lung was either solid or a cavity and the pleural cavity was smaller than the intrapulmonary, the signs of which were only too evident and the development of which could be followed while in the ward. The tuberculous process was rather acute, death ensuing about nine months from onset.

CASE 19.—*Partial pyopneumothorax at the right base, partial circumscribed pneumothorax at the right apex, both due to phthisis with cavity formation; death; autopsy.*

James M. Autopsy No. 1293.

ANATOMICAL DIAGNOSIS.—*Chronic pulmonary tuberculosis; confluent miliary tubercles. Gelatinous pneumonia. Fibroid induration. Cavity formation. Perforation of cavity and pleura. Pneumothorax. Perforation of cavity through pleura into œsophagus. Tuberculous ulcers of intestine. Gangrenous cavity in lung. Pulmonary œdema.*

Length of body 169 cm. Thin colored man.

Left lung is free from adhesions. Right lung at the apex is densely bound to the pleura. The lower portion of the pleural cavity contains a considerable quantity of yellow fluid, containing fibrinous masses. On incision into the pleura there was an escape with hissing sound of foul-smelling gas.

Left lung is voluminous. Surface is smooth. On section lung is of a grey-red color, deeper red below. Lung is very œdematous. Throughout the lung are discrete and confluent tubercles, usually collected into groups of split pea size. These are less abundant in the lower lobe.

Right Lung.—For a distance of 10 cm. from the apex the lung is densely adherent to the parietal pleura. Below this point the cavity is not obliterated but is distended with brown fluid containing soft brown blood-clots; gas, as mentioned, escaped on incision. In this region the walls of the cavity are increased in thickness, measuring 1.5 mm. The surface of both parietal and visceral pleura is covered with a layer of opaque, white, tough, fibrinous material. On the outer surface of the lower lobe, opening into the pleural cavity, is a round orifice, 12 mm. in diameter, communicating with a small cavity in the lung substance below. The edges of this opening are rounded. On the inner side of the upper lobe, near the apex, is a small cavity of cleft-like form, between the lung surface and the trachea, immediately above the bifurcation; it measures 3.5 cm. anteroposteriorly; 2 cm. from above down. This represents the only indication of pleural cavity in this region; elsewhere there is complete obliteration. This cavity communicates by two openings with the series of large cavities in the lung substance, which here approach the surface closely. The anterior opening is 1 cm. in diameter; its edges are rounded; a posterior

one is narrow and puckered. The cavity sends a projection in front of the trachea immediately above the bifurcation as far as the middle line. A second prolongation extends behind the trachea obliquely upward for a short distance and opens into the œsophagus at a point 6 cm. below the cricoid cartilage, 18 cm. above the cardiac orifice. The walls of the cavity are smooth and of greyish fibrous appearance. The upper lobe of the lung is riddled by very irregular communicating cavities which almost completely replace the lung substance. The largest is situated almost immediately below the pleura of the outer surface and measures 9 x 7 x 6 cm. It is crossed by numerous vessels which have been dissected free. The tissue which remains is densely fibrous, particularly near the root of the lung. Near the anterior margin at the lower part of the lobe is tissue of grey gelatinous appearance with here and there small, opaque, yellow areas. The lower and middle lobes are thickly studded with conglomerate tubercles, partially caseous. At the extreme base is a cavity about 2 cm. in diameter with soft irregular walls of necrotic appearance. The cavity which opens on the pleural surface measures 2 x 1 cm. and communicates with a medium sized bronchus.

This was not one of the ward cases, hence a clinical history is wanting.

It is of interest that at autopsy the foul-smelling gas hissed from the right pleural cavity. The pneumothorax was partial, the upper lobe being in good part adherent. The perforation was large, 12 mm. in diameter, on the outer surface of the lower lobe and opening into a small cavity. At the apex of the pleural cavity, however, is a second small pneumothorax cavity which opens into the œsophagus.

CASE 20.—*Hydropneumothorax due to tuberculosis; recovery. Pyopneumothorax due to phthisis with cavity formation.*

John D., aged 25, white (1893).

First admission July 11, 1893. **Med. No. 3087.**

Discharged September 11, 1893.

Clinical Diagnosis.—*Pulmonary tuberculosis; pneumohydrothorax.*

He complained of pain in the left side and shortness of breath.

Family History.—One sister died of tuberculosis.

Past History.—He was very healthy when young, present illness being his first serious sickness. Habits are good. Occupation: a steam pipe fitter.

Present Illness.—He has been troubled with shortness of breath for seven months. This became severe three weeks ago. He is unable to lie down. The pain is dull and increased by breathing. His feet and ankles were swollen one week ago for the first time. He has no cough; no expectoration. He has had fever and sweating; no chills. He stopped work one month ago.

Physical Examination.—Respirations are 44 to the minute. On inspiration the left side of the chest is more prominent than the right. The apex beat is diffuse in the 3rd, 4th and 5th spaces on the right side. The slightest exertion causes great dyspnœa. Behind is pronounced bulging of the left side. On percussion in front in the recumbent posture the resonance extends to the 8th rib in the mid-axillary line. In the posterior axillary line there is dulness in the 6th space. Cardiac dulness obliterated.

Right side clear.

Backs.—At the right apex the note is slightly dull as low as the spine of the scapula; below is clear. The left side is clear almost to the base. There is, however, well marked dulness at the base and absent fremitus.

Auscultation.—On the right side, clear; on the left it is enfeebled, especially after coughing. Behind breathing is feeble until one reaches the angle of the scapula where it has a marked amphoric quality. The cough has a metallic quality. The breath sounds in the lower back are distant. The succussion splash is well heard. The movable dulness is marked.

Heart sounds are clear.

Abdomen negative. The coin sound on the left side is distinct. The aspirating needle showed on the left side a serous effusion. The fluid escaped under considerable pressure. The patient afterwards was more comfortable. The sputum is scanty; no tubercle bacilli are found.

Urine.—A faint trace of albumin.

July 15. Tubercle bacilli are present in the sputum.

August 12. There is marked fulness on the left side of the chest. The apex beat is below the tip of the ensiform. The cardiac dulness extends 3 to 4 cm. to the right of the nipple line. In the recumbent posture in the anterior axillary line resonance extends as low as the 8th rib. It is of a somewhat wooden tympanitic quality. In the erect posture flatness begins at the 6th rib. Resonance extends as low as the 11th rib behind. Resonance throughout the right side is good. In the left front, respiration has a distinct amphoric quality, and on coughing or moving there is distinct metallic tinkling; succussion is well marked.

September 2. Dr. Osler: Over the left back breath sounds are feeble and distant. Inspiration less intense than expiration and without any special amphoric quality. Expiration is prolonged, markedly amphoric. It gives one the impression of a much larger volume of air. Coin sound is well heard. No special movable dulness.

September 11 the patient left the hospital.

Second admission September 20, 1893. **Med. No. 3301.**

Discharged September 28, 1893.

Diagnosis.—*Hydropneumothorax; pulmonary tuberculosis.*

Present Condition.—The patient left the hospital and was well until six days ago when he began to suffer from dyspnœa. No cough; no pain in the side. He has some pain in the stomach.

Physical Examination.—There is some dyspnœa; some cyanosis; respiration 32; pulse 88.

Thorax.—The left side is not so distended as in the previous admission. The respiratory movement is limited. The cardiac impulse is diffuse just to the left of the sternum. It is more forcible to the right of the sternum.

Percussion.—There is very slight difference between the two sides above. The left is a little more tympanitic. In the recumbent posture there is no dulness in the front. In the sitting posture dulness reaches the lower border of the 3rd rib and extends to the base. The right side is clear throughout in front and behind. The left back is clear to the angle of the scapula, below which it is dull with absent fremitus.

Auscultation.—The right side is clear with a few moist râles. On the left side behind breathing is very much enfeebled, almost absent at the base. The amphoric quality is heard slightly after coughing when there are numerous râles, some metallic. The coin sound is obtained just inside and above the angle of the left scapula. Succussion splash is well heard. On auscultation in the front the breath sounds are diminished in intensity over the entire left front, the amphoric quality being only slightly heard after deep inspiration. There are no metallic râles.

Heart.—The relative dulness extends half way to the right nipple. The apex by auscultation seems to be just below the ensiform cartilage. The sounds are everywhere clear. The liver is not depressed. The abdomen is natural.

September 24. To-day the dulness is not quite so high; amphoric quality is well heard in the front and behind. The coin sound and the Hippocratic succussion are more distinct.

September 28. The patient is discharged to-day. The fluid in the chest appears of about the same amount as on admission. Flatness begins in the 3rd space in front in the erect posture. All the phenomena of pneumothorax present. The patient has had no fever since admission. The cardiac dulness extends only 2 cm. to the right of the sternum; the apex beat is in the epigastrium.

Third admission October 5, 1893. **Med. No. 3364.**

Discharged October 14, 1893.

DIAGNOSIS.—*Hydropneumothorax; pulmonary tuberculosis.*

Admitted COMPLAINING of increased shortness of breath and feeling of general weakness. Since discharged last time he has been getting slowly worse. The attacks of coughing are severe.

PHYSICAL EXAMINATION shows slight immobility with fulness of the intercostal spaces. The cardiac impulse is in the epigastrium and extends to the right of the sternum in the 3rd and 4th i. s. Relative resonance to the 5th rib on the left and shading there into a dulness which is movable. Behind the dulness, below the angle of the scapula, breathing is feeble. Signs of pneumothorax not present on auscultation. The breath sounds are distant; nowhere have they amphoric quality even on coughing. The coin sound is not present. With the patient erect there is one spot in the upper axilla just under the pectoral fold where is slight amphoric resonance.

October 12. Dulness begins at the 2nd rib in the nipple line. The note is perfectly flat below the third. Respiratory murmur is much diminished over this area. Feeble tactile fremitus. Behind dulness begins at the angle of the scapula.

October 13. Patient aspirated. 1000 cc. of yellow, somewhat turbid fluid was removed.

October 14. The right side of the chest moves more than the left. intercostal spaces are obliterated. The heart's impulse is chiefly in the epigastric region. There is high pitched Skodaic resonance down the right front. There is no dulness until the lower axilla is reached in the recumbent posture. On sitting up the note is dull at the apex, flat at the third rib which extends to the base. On auscultation breathing is somewhat amphoric and the râles have a very marked metallic quality. There is deficient resonance behind, extending to the angle of the scapula. The amphoric breathing is not well heard behind. No râles except on coughing, and there is distant breathing and somewhat deficient tactile fremitus at the base. The coin sound is not present. Succussion still well marked.

October 17. The patient leaves the hospital improved. The temperature was slightly elevated for four days since admission.

Fourth admission February 1, 1894. Med. No. 3673.

Discharged March 10, 1894.

Clinical Diagnosis.—*Pneumohydrothorax; pulmonary tuberculosis.*

The patient since leaving the hospital remained fairly well until two weeks ago when shortness of breath began to return. This has been his chief complaint. He has had little cough.

Physical Examination.—The urine has a faint trace of albumin. The patient looks well; color good. The movement of the chest is less on the left side, particularly at the base. The intercostal spaces not so marked as on the right side. There is no distinct apex beat. Marked pulsation at the xyphoid angle; also seen in the 3rd left interspace and feebly in the 4th right space. No dyspnœa.

Percussion.—Dulness everywhere from the clavicle down on the left side. The note is flat without a tympanitic quality. The resonance from the right lung extends across the manubrium. There is flatness from the apex to the base behind; tactile fremitus not completely absent.

Auscultation.—In the left supraclavicular region the sounds are loud, somewhat cavernous; expiration prolonged. From the spine of the scapula down there is feeble respiration, scarcely audible below. In the interscapular region close to the spine is loud, somewhat tubular breathing. On coughing there are coarse râles in the supraspinous fossa and over the rest of the back distant mucous râles. "It is interesting that there should be so many râles in the back." Breathing over the right side is loud and clear. Numerous moist râles over the right supraspinous fossa. In front there are loud, clear breath sounds on the right side. In the left infraclavicular region distant tubular breathing with an occasional râle. This is heard as low as the nipple. In the axilla the breath sounds are tubular, distant; no amphoric sounds; no succussion splash; no coin sound.

February 9. The patient was aspirated and 650 cc. removed. Cultures from the aspiration negative. Sputum contains a few tubercle bacilli.

March 1. Patient aspirated; 240 cc. of a cloudy, yellow fluid obtained. There is no succussion splash; no coin sound. The point of maximum impulse is not sharply made out. There is apparently no dulness whatever to the right of the sternum; sounds are best heard in the 5th interspace about 5 cm. inside the mammillary line.

March 5. For the last three or four days the patient has been coughing at intervals, small amounts of blood. Never more than 1 drachm at a time.

On the 10th of March, at his own request, he is discharged.

Temperature during this admission was irregular, reaching from normal to 103, gradually falling but remaining irregular until dismissal.

Surgical No. 3155.

Fifth admission to the surgical side June 10, 1894.

Discharged August 22.

DIAGNOSIS.—*Empyema; tuberculosis of the left side; pyopneumothorax of long standing.*

OPERATION June 11, 1894. His 7th rib was excised in the mid-axillary line. The lung was entirely collapsed and an opening in the lung admitting air was felt in the lower portion. The heart could not be felt. The cavity was filled with a thin, yellow, odorless fluid. It contained large masses of yellow tissue, probably fibrin; no blood. The cavity was evacuated and drained. The patient improved and was discharged August 4. The wound is still deep and discharging pus but his general condition is good. He was readmitted twice later. The first time September 23, 1894, and again October 2, 1894. On this latter occasion there is an accumulation of thick, curdy pus in the pleural cavity. The patient is much emaciated. He died May 17, 1895.

The operator drew a diagram of the lung as felt at operation. The perforation was on the lateral surface of the lower lobe.

This was a case of pleurisy with effusion and pneumothorax not, so far as we know, due to aspiration. On admission every sign nearly of pneumothorax was present except amphoric respiration. The breath sounds were feeble and amphoric only on expiration. The succussion splash, coin sound, metallic tinkling, hyperresonance and displacement of the heart were all present. Eight months later (March, 1894) every one of these signs was absent. Hence we may consider the pneumothorax cured.

The pneumothorax found at operation probably means another attack.

CASE 21.—*Pyopneumothorax following empyema; operation; recovery.*
William K., white, 20 years old. **Med. No. 738.**

Admitted September 19, 1890. **Surg. No. 651.**

Transferred to surgical side October 2, 1890.

Operated on October 2, 1890.

Discharged November 28, 1890.

Clinical Diagnosis.—*Pyopneumothorax.*

Complained of "dyspnœa, cough, loss of flesh, sweating."

Family History.—Good; no lung trouble.

Past History.—The patient was always well until last June, 1890, when he had "malaria."

Present Illness.—In July he had the "symptoms of typhoid fever." He says he was recovering when, on July 23, he had a "stitch in the right side," and this was followed by cough, renewal of fever and signs of pleurisy with effusion. He has had no expectoration at any time. He has had night sweats; is losing considerable flesh. Three weeks ago a physician aspirated about three pints of pus from the chest. This was followed by a week of relief but since then the symptoms have increased in severity.

Status præsens.—Emaciated. Temperature, 103°, pulse 96, respiration 24. He lies on the right side. The temperature is irregular; there is much sweating.

Thorax.—The right side is absolutely immobile, is flatter and looks smaller than the left. There is a general fulness in the lower axillary and hepatic region. At the base tactile fremitus is absent. On percussion with the patient flat on his back the whole front from clavicle to the upper margin of the rib gives a full tone with slightly tympanitic quality, but in the erect posture the absolute dulness reaches the 3rd interspace and above is Skodaic resonance. Auscultation of the resonant area gives very loud amphoric breath sounds and râles with a distinct metallic ring, and on coughing râles with a characteristic amphoric quality.

Left side.—Nothing abnormal.

Liver not palpable.

Urine.—Negative.

October 2. The patient has gained strength; the fluid has not increased.

Operation.—Dr. Halsted: The eighth rib was resected; about 1000 ccs. of creamy pus evacuated. The patient improved rapidly. The lung in about two months had expanded to fill the pleural cavity. January 15, 1895, he wrote that he was in perfect health.

In this case right-sided empyema developed and was aspirated six weeks after the onset of the pleurisy. The patient was admitted to this hospital three weeks after the aspiration and the diagnosis of pyopneumothorax made from the hyperresonant percussion note, the extreme movable dulness, the amphoric respiration and the metallic tinkling. Such movable dulness as this, the tone resonant to the costal margin in recumbent posture and flat from the third rib down in the erect, must mean pyopneumothorax.

The cause of the condition is in doubt. Surely it was not the rupture of the empyema through the lung since the patient never had any sputum. It may have been the rupture of a small tuberculous cavity, but here also the patient was aspirated previous to admission which may explain the condition.

CASE 22.—*Right-sided pyopneumothorax due to tuberculosis; spontaneous recovery; recurrence; death; autopsy.*

Christian T., white, age (1889) 39.

First admission September 25, 1889. **Med. No. 159.**

Discharged December 19, 1889.

DIAGNOSIS.—*Tuberculous pleurisy.*

FAMILY HISTORY.—Is tuberculous. His wife died of tuberculosis.

PAST HISTORY.—He has had the diseases of childhood and later chills and fever; no other diseases. Has been a healthy man. His business has been a waiter and bartender.

PRESENT ILLNESS began January of this year with shortness of breath after walking or upon any exertion. There was no pain in the chest or any part of the body; no swelling of the legs. In January he had chilly feelings and severe headaches for two days. Then began cough with pain. One year ago he had a bad cough with much expectoration, at one time spitting up a mouthful of blood and followed by blood-stained sputum for two days. During January he lost much flesh; had no pain on breathing; no appetite; became very weak. This lasted, with much shortness of breath, for two weeks. The shortness of breath has continued to date.

PHYSICAL EXAMINATION.—The patient is a large, well built man. Respiration quiet; temperature normal; pulse 72. The chest is well shaped. Expansion equal on quiet respiration but diminished on the left on deep inspiration. The apex beat is visible in the 5th space 1½ cm. outside of the nipple line. When sitting up the apex beat reaches almost to the axilla. Behind the right side looks a little fuller than the left. Tactile fremitus is absent over the right side and absolute dulness extending in front to the left border of the sternum.

Auscultation.—On the right back there is silence. Towards the spine there is tubular distant breathing. In front feeble distant breath sounds on the right.

September 29. Cardiac dulness is two fingers' breath beyond the nipple line. On aspiration 30 ounces of fluid were removed.

October 1. 30 ounces of fluid again removed. Dulness begins at the right edge of the sternum.

On the 6th aspirated again, 21 ounces obtained.

October 18. 32 ounces. After aspiration the percussion note is resonant to the 3rd interspace.

October 25. In recumbent posture the note on the right subclavicular region is clear; dulness begins at the nipple. Posteriorly it is absolutely flat.

December 19. The patient since the last note has been aspirated three times. At present he is in good condition, has some shortness of breath when walking fast. Has cough and expectoration. The sputum has been examined repeatedly for tubercle bacilli which, however, have not been found.

Right lung.—Clear as far as the 3rd rib. Below this there is absolute flatness on percussion. In the upper axilla the resonance is impaired; below it is flat. Behind it is high pitched to the spine of the scapula and below is flat. The left lung is clear. On auscultation of the right side breathing is tubular in the upper lobe with bronchophony and pectoriloquy over the rest of the lung. Breath sounds are entirely absent. Fremitus is absent. Patient discharged.

Temperature while in the hospital varied from 98-99.5 rising at one point (October 24) to 102 after aspiration.

Second admission May 28, 1896. **Med. No. 6160.**

Transferred to the surgical side August 4.

Clinical Diagnosis.—Empyema, pneumothorax, pulmonary tuberculosis. He complained of cough, pain in the side, shortness of breath, loss of voice.

Present Illness.—Since leaving hospital he has felt well and was able to work regularly until one week ago. Ten weeks ago he began to feel badly; coughing and shortness of breath. He continued to work until one week ago, although his condition was gradually getting worse. He has not felt much pain. The cough, shortness of breath and weakness are his chief symptoms. The appetite very poor.

Physical Examination (Dr. Osler).—Face somewhat flushed; pulse is good. The chest is fairly symmetrical. The right clavicle is not more prominent than the left. The shoulder is not depressed. There is fair expansion on both sides but much more on the left than on the right. The resonance affected from the clavicle to the 3rd rib on the right side. At the 3rd rib it shades into absolute flatness. There is flatness behind from the spine of the scapula down. The sputum: No tubercle bacilli were found; there are, however, elastic fibers.

June 2. This morning the patient is very cyanosed; pulse 144; soft and full. The patient lies quietly on his right side. Cardiac impulse is not distinctly marked. The dulness is entirely covered by pulmonary resonance. The sounds are best heard in the 5th space just outside the nipple. There is marked dulness throughout the right front, nowhere complete flatness. Respiration over the dull area is feeble and distant. Temperature 103.4°. There is some dulness well marked in the back; nowhere complete flatness above the 8th rib. There is nowhere any cardiac murmur. Over the right front are medium moist râles. Along the right border of the sternum respiration has a distinct, somewhat amphoric quality, especially in the second right space close to the sternum. Throat examination: There is adductor paralysis of the left vocal cord. Muscles of the left side of the larynx are much wasted.

During the night of the 2nd and 3rd of this month the patient improved markedly and yesterday passed a fairly comfortable day. This morning he is lying on his back, still somewhat cyanotic. Respirations ranged during the last twenty-four hours from 20 to 28 per minute. This morning the upper right chest is flattened. The whole side moves less on inspiration. The point of maximum impulse is not made out on palpation. Over the entire right upper front there is well marked tympany which becomes flat at the 7th rib in the mammillary line. The posterior right side looks fuller than the left, although it moves a little. The same tympanitic note is to be made out in the back, although it is duller than in front. In the erect posture there is flatness at the 5th rib in the mammillary line; movable dulness therefore amounting to 10 cm. On auscultation there are fine moist and sibilant râles from the apex to the third rib, increased on coughing. Above the right clavicle the breath sounds are somewhat amphoric; below this they have a rather indistinct tubular quality with numerous coarse râles on inspiration and sibilant on expiration. They are heard, although faintly, until one reaches the flat area. Throughout the right back respiration is distant and indistinct; below the point of flatness entirely absent. The coin sound is not well heard. The succussion sound is distinctly heard and the patient says he hears something splash. The heart sounds are best heard in the 6th space outside the nipple line and 15 cm. from the mid-line. The heart has therefore moved to the left.

Dr. Osler: June 10. In the erect posture the note is full, not tympanitic in the 1st and 2nd spaces on the right side. On auscultation the breath sounds are nowhere amphoric. Moist râles of medium size, not metallic heard over the right front. Below and on the 6th space, particularly inside the nipple, inspiration and expiration become slightly amphoric, more so than before and in the 6th interspace in the axillary line on deep inspiration they have an amphoric quality, and an occasional metallic râle. Litten's sign not seen on either side. Below, on auscultation, the most striking feature is the distant feeble character of the breath sounds; nowhere amphoric phenomena, either on deep breathing, not even on coughing. Sputum contains a few tubercle bacilli; no elastic fibers to-day.

June 17. Dr. Osler: In the erect posture the Skodaic resonance is well marked; breath sounds are a little amphoric in this region. In the recumbent posture the resonance on the right side reaches the 6th space; in the erect the 3rd space. Succussion is still present. Coin sound is not well marked.

August 3. The temperature since the last note has remained perfectly normal; hitherto it has varied from 97 to 101. He has gained nine pounds since admission and feels well enough to go. This morning flatness begins at the 4th rib in the erect posture on the right side, 5th in the recumbent posture. In the back, however, there is flatness nearly to the spine of the scapula. The cardiac impulse cannot be sharply made out but the sounds are best heard in the 5th and 6th spaces just outside the nipple line. On auscultation, respiration is feeble and indefinite at the

apex but no longer amphoric. The coin sound is not present. Exploratory puncture shows a yellowish-green purulent fluid, containing fat droplets and cholesterin crystals.

This case is of much interest on account of the spontaneous cure of the pneumothorax occurring under observation.

Transferred to the surgical department August 4, 1896.

Surgical No. 5830.

Operation August 5.

Discharged October 23, 1896.

OPERATION.—Dr. Finney: Resection of the 8th rib and thoracotomy. Drainage of the chest.

About 1500 cc. of pus were found in the cavity. The patient did well, the pleural cavity drained well and was daily irrigated with warm boric solution. The cough was considerable and expectoration large in amount. Improvement continued till October 18, when the cavity was reduced to a sinus holding only about 300 cc. of fluid.

The temperature till August 11 ranged from normal to 102°, and thereafter seldom rose to 101°.

January 18, 1897. The patient was seen by Dr. Osler, who makes the following note: "He looks badly; great deal of shrinkage on the right side; dulness over the right back; fremitus is felt except at the extreme base, but is enfeebled. At the right apex behind, the breath sounds are feeble, distant, a little tubular on deep inspiration and on coughing. At the right interscapular region inspiration and expiration are distant, tubular and scarcely audible. In the front is a great amount of shrinkage. There is flatness on the right side in front, very feeble distant breath sounds. The heart sounds are louder transversely. In the upper axilla auscultation is almost silent. Moist sounds at left apex."

Fourth admission June 21, 1897. **Surg. No. 6657.**

Discharged July 10, 1897.

Since the operation he has felt fairly well and has gained in weight. He has had occasional cough and expectoration and shortness of breath, which last is his chief symptom. The discharge from the wound varies in amount. The sinus is very small.

Operation deemed inadvisable.

Fifth admission June 16, 1898, age 48. **Med. No. 8688.**

Died June 24.

CLINICAL DIAGNOSIS.—*Pyopneumothorax; pulmonary tuberculosis.*

PRESENT ILLNESS.—He was discharged October 23, returning for irrigations until March, 1897. In August he had a severe coughing spell which ruptured the scar and there was a discharge of pus; wound healed. He has had a great deal of coughing and expectoration during all this time. The expectoration is purulent, not bloody. He felt so weak and helpless lately that he could not walk. Is very short of breath. Pain across the abdomen on exertion. The legs are much swollen.

PHYSICAL EXAMINATION.—The right clavicle is prominent. The infra-

clavicular space much depressed. The left thorax is much more prominent than the right. Respiratory movements are almost confined to the left side. The point of cardiac impulse is not well seen or felt but is present in the 5th and 6th spaces 14 cm. from the median line. Over the left pulmonary area in front, back and axilla the note is somewhat hyperresonant. Over the whole right lung the resonance is impaired and modified. At the apex in front there is distinct dulness of resonance and in the infra-clavicular region there begins a high pitched Skodaic tympany which extends downward into the axilla. At the base the note loses its tympanitic quality. Similar relations exist in the back. The most tympanitic note is heard below the spine of the scapula.

Auscultation.—Left side. Over the back and front the respiratory sound is vesicular. Over the upper front there is an occasional sibilant râle. Over the right back the breath sounds at the apex are tubular and expiration somewhat prolonged. Just below the apex the sounds while tubular are echoing and amphoric, accompanied by numerous fine metallic râles. They are heard around into the axilla. Toward the base posteriorly the amphoric quality is lost and the breathing has a more vesicular character with moist râles over the front of the chest. Right front: At the extreme apex breathing is harsh and tubular. Just below the clavicle the sound becomes echoing and metallic with occasional tinkling râles on inspiration, increased by coughing. This type of sound is heard down to the level of the 3rd space in the parasternal line and to the 5th space in the anterior axillary line. Below this oblique line the breath sounds are harsh with crackling inspiratory râles. Over the whole right side of the chest the breath sounds are distant and suppressed. In the area of amphoric respiration there is whispered pectoriloquy. The voice sounds are muffled and distant. In the mid-axillary line they have a distinctly metallic sound.

There is well marked œdema of the ankles. Liver dulness begins at the 6th space in the nipple line and extends 5 cm. below the costal margin.

June 18. Dr. Osler: Intense accentuation of the pulmonic second; loud amphoric breathing over the right front. The right shoulder is depressed. The clavicle is prominent. The right base is flattened. The patient is somewhat cyanotic. The heart's impulse is rapid; respiration labored and grunting.

June 24. This morning the patient became very much cyanosed, breathing is irregular and gasping; heart rapid and irregular. He became drowsy and died at noon.

The urine contained no albumin, an occasional hyaline cast. The temperature was 99 from admission to the 20th inst. and 96 at the time of death.

Autopsy No. 1110.

Anatomical Diagnosis.—*Chronic adhesive and proliferative pleuritis. Empyema and pneumothorax. Great compression of right lung. Thrombosis of right pulmonary artery. Tuberculous cavity, left lung. Disseminated, chronic conglomerate tuberculosis, this lung. Chronic adhesive me-*

diastinitis. Chronic adhesions about trachea and aorta. Congestion of liver, spleen and kidneys.

Body of a large, strongly built man. No subcutaneous œdema. Rigor mortis.

Diaphragm opened under water on the right side, permits a few bubbles of air to escape. On the left side none escapes.

Left lung is voluminous and free from adhesions. On section it presents a coal pigmented and mottled appearance. Scattered over the cut section are numerous areas of conglomerated and partially caseous tuberculous masses. Between these the lung tissue is congested, but air-containing. The pleural surface of the lung is pigmented and the nodular masses can easily be felt beneath the surface. The bronchi contain muco-pus, the mucous membrane is congested. The blood-vessels are free. In the apex of the lung there is a cavity the size of a walnut, possessing a distinct capsule lined with caseous material and containing caseous, broken-down matter. The consistence of the lung is increased.

Right lung.—The right pleural cavity is converted into a sac with board-like walls. On attempting to remove the lung with the parietal pleura, this latter membrane is found to be dense and board-like and comes away only with the aid of much dissecting from the ribs. The thickened membrane also includes the diaphragmatic part of the pleura. On removing these parts pus wells up from the cavity. This pus is thin and of typical yellowish-grey color and is not frothy. Its quantity cannot be estimated, but not less than a liter and probably more, escapes. After removal the greatest thickening is found in the parietal pleura. The lung itself is greatly compressed; it occupies the upper portion only of the pleural cavity; its pleura is also though much less thickened, white in color, puckered, with here and there deeper depressions. Actual communication with the lung substance from which the air escaped into the pleura, not discovered. The bronchi are free. The main branch of the pulmonary artery contains an adherent, mixed, thrombus.

This patient at the time of his second admission gives a history of tuberculosis of the lungs covering at least $6\frac{1}{2}$ years. During the first admission he was aspirated seven times and was sent out very much improved and able to work for nearly five and a half years. At the time of his second admission he gave a history of cough, shortness of breath of about ten weeks' duration. Fluid was found in the right chest. The pneumothorax developed between June 3 and June 10, but as the note is undated we cannot state the exact date. The patient was found cyanotic. The percussion note over the right side was full with movable dulness of 10 cm. The succussion splash was easily obtained. A remarkable feature of the case were the distant respiratory sounds. No metallic phenomena, no coin sound were given. The pneumothorax disappeared spon-

taneously while the patient was under observation in about two months.

The empyema was operated on and the patient improved, but the intrapleural trouble continued although the wound healed. On his last admission nearly nine years after his first, he presented the signs of pyopneumothorax. In August of the preceding year, ten months before, the sinus had again opened but also healed. It is possible that the air which entered at that time was that found on admission and demonstrated by the water test at autopsy. Over the whole right lung resonance is impaired, but this would be expected for the pleura was board-like. Over the lung greatly compressed at the apex the note was dull, below it was of Skodaic tympany. Over the whole right front there was, June 18, loud amphoric breathing, interesting since the fistula was probably not an open one, at least at the autopsy could not be found.

Résumé.—These 22 cases were quite certainly due to tuberculosis. How many more were we cannot say, for pleurisy is now recognized as very often of tuberculous origin.

It is impossible to state what percentage these represent of tuberculosis cases, because the hospital refuses the majority of such patients, but the autopsies on cases of tuberculous pneumothorax represent just 4 per cent of the cases of tuberculosis of the lungs which came to autopsy. In this total are included all cases with a clearly tuberculous lesion of the lung, acute or chronic, a lesion pronounced enough to be mentioned in the anatomical diagnosis. It is hard to tell just where to draw the line between a tuberculosis in acute or chronic stage, but of those in which the lung lesions were manifestly acute the pneumothorax represent just about 8 per cent.

Sex.—Of our cases 18 were men, and 4 women. The ages were distributed as follows: Third decade, 8; fourth, 7; fifth, 4; sixth, 2; not stated, 1.

There seems no preference for sides, 11 were on the right and 11 on the left.

Of the 19 cases with autopsy, the pneumothorax was complete in 15, circumscribed in 4.

The perforation was found in 16 cases: in 7 in the upper lobe, 6 in the lower, and 3 in the middle. In 7 cases it was 3 mm. or less in diameter, in 5 above 1 cm. It is of interest that of these large fistulæ the duration of pneumothorax before death was in one case 10 days,

in another 3 months. In one case of but 15 hours' duration the fistula measured 6 x 3 mm. In one case there were two large fistulæ, one 10 and one 12 mm. in diameter.

As regards the duration of tuberculosis before the onset of the pneumothorax, the shortest was 4 weeks, the longest over 12 years. Five cases were of over 1 year's duration, 8 of between 6 months and 1 year.

But two of the 22 cases left the hospital, and they were cases in which the symptoms of empyema predominated. We do not know how long they lived.

Of the 19 cases with autopsies there was CAVITY FORMATION in all but one (No. 12) and in No. 2 without autopsy the symptoms showed their presence.

Of 18 cases the *fluid* was purulent in 14, serous in 1 and absent in 3.

Other notes on the pathological findings of these cases are included in the discussion of the physical signs.

Bronchiectasis.—It is remarkable that so few cases of pneumothorax are on record as due to this disease. Biach in his long series could find but ten cases and Gebrak[345] could only add one to that number. This was the case also reported by Letulle.[344] In this case a tuberculous ulceration perforated the wall of the bronchiectatic cavity. The case is also interesting because it occurred in diabetes, a condition commonly associated with tuberculosis, but perhaps in no other case with pneumothorax. Among the cases reported are those of Hotte,[92] Rumpf,[96] in which case the sequence was pneumonia, bronchiectasis, gangrene, and West's case[104] due to chronic bronchitis leading to bronchiectasis.

We would add to these eleven cases the following two:

CASE 23.—*Right-sided pyopneumothorax due to bronchiectasis; operation; death; autopsy.*

John T., aged 34, colored. **Med. No. 3170.**

The patient was admitted August 9, 1893, and transferred to the surgical side August 19, 1893. He COMPLAINED of shortness of breath and pain in the right back.

The mother died of tuberculosis.

PAST HISTORY.—The patient has been a healthy man. He has worked as a stevedore. Ten years ago he was in the hospital for ten months, disease not stated. Following this his abdomen was swollen. Early this

year he had an attack of pneumonia; was sick three months and has not been well since. He has been suffering from cough with a yellowish sputum. He has night sweats and fever, pain in the right side, especially on exertion. He has worked hard until six weeks ago.

PHYSICAL EXAMINATION.—Sputum contains no tubercle bacilli. The blood shows a marked leucocytosis. The urine contains a trace of albumin, a few granular casts. There is slight arterio-sclerosis. The chest examination was negative.

August 13. The temperature has been gradually rising, reaching to-day 103. With this rise in temperature respirations increased to 32. The pain in the right side has increased, especially on deep breathing. There is orthopnœa. Over the right base and axilla there is increased fremitus with moist râles. The percussion note is higher pitched.

August 15. There is dulness on the right side to the 5th rib in the mammillary line. There is no fremitus over this region and feeble breath sounds. Motion is diminished.

August 19. Area of dulness has increased. Patient complains of much pain.

Transferred August 19. **Surgical number 2427.** OPERATION, Dr. Finney. A needle introduced into the posterior axillary line at the 7th rib obtained no pus, but when introduced 2 cm. posterior to this line over the 8th rib pus was obtained. The 8th rib was resected in this place and a cavity found extending upwards and towards the spine. The diaphragm could be felt below. Four ounces of fluid were removed. The wound drained. The patient improved.

September 20. Dr. Osler examined the chest and pronounced both lungs in good condition. The wound has healed.

Second admission October 1, 1894.

Transferred to the surgical side March 30, 1895.

The patient has been on the medical side since October, 1894, for a bronchiectatic cavity. The note made to-day by Dr. Thayer is as follows: Resonance good everywhere but in the right axilla. The breath sounds are good except just inside the scar. There are no adventitious sounds.

Fourth admission October 11, 1895. **Med. No. 5454.**

Discharged November 4, 1895.

CLINICAL DIAGNOSIS.—*Bronchiectasis.* Two weeks ago the patient was attacked with night sweats, cough and shortness of breath. This was so severe that he could not sleep but must sit up in bed. About one week ago his feet and legs were swollen. An offensive expectoration was associated with the cough. This has been present since August, 1893, but not to the extent or the amount as at present. The pain was so severe that on October 7 the patient had to go to bed and would cry out because of it. The sputum was very offensive, so much so that it nauseated him. It has been blood-tinged.

PHYSICAL EXAMINATION.—The chest moves well on inspiration on both sides. At the right lower half is slightly deficient expansion. There is

slightly deficient resonance below the spine of the scapula; flatness below the scar. The vocal fremitus is present but diminished throughout the dull area. In front resonance is a little exaggerated, the flatness reaching to the mid-axilla. The left side is clear. The right side: Respiratory sounds are clear above and become enfeebled as one passes downward into the dull area. There are no adventitious sounds. The urine is negative. Temperature practically normal.

Fifth admission November 23, 1895. **Med. No. 5586.** Transferred to the surgical side March 4, 1896.

Clinical Diagnosis.—*Bronchiectasis, chronic pleurisy, pneumothorax.*

The patient complains of shortness of breath and general weakness. He returned to-day in a very weak condition. Shortly after his arrival in the ward he had a hæmorrhage, expectorating about half pint of bright red blood. This evening he is propped up in bed; temperature 99.4; pulse 128; respirations 40.

November 24. The patient has orthopnœa. The general condition is the same as on last entry. The breath is foul. The thorax appears symmetrical. Expansion is more marked in the lower left front than on the right. There is well marked dulness in the right axilla below the 5th rib. Vocal fremitus is diminished throughout, although it can be felt. In the dull area in the right axilla respiration has a distinct, somewhat tubular modification, especially in the upper part, though it is free from adventitious sounds. Resonance is good in the upper back; somewhat deficient in the right back near the angle of the scapula. The note is flat below the scar. In the right back between the 10th and the 7th ribs there is a small area where there is distant tubular respiration with the voice sounds markedly increased. There are no adventitious sounds heard after coughing. The left back is clear.

November 30. The patient passed a very uncomfortable night, coughing a great deal and expectorating great quantities of sputum which is always blood-tinged.

December 3. Dr. Osler: The patient is feeling more comfortable.

February 14. The dyspnœa and general distress of the patient are out of proportion to his general appearance. Respirations 40. Flatness on the right side to the clavicle also over the back. Tactile fremitus is diminished but not quite absent. In the middle of the lung there is a distant tubular breathing at the apex and the breath sounds are feeble and scarcely audible at the base. There is puffy tubular expiration at the angle of the scapula. The dulness crosses the midsternal line. The apex beat is in the 6th space in the axillary line. The liver is a little depressed. The right side of the chest appears a little large. The intercostal spaces on the right side are not so well marked. There is flatness from the clavicle down. The right side measures 1¼ inches more than the left.

February 10. 1600 cc. of fluid were removed from the right chest. The note became Skodaic to the 4th rib on the right side.

February 15. There is flatness at the 4th rib and throughout the axilla.

Over the right upper front there is Skodaic resonance. There are diminished breath sounds, prolonged expiration over the upper front and a few crackling râles. Below the 4th rib in front breath sounds are absent. Throughout the axilla they are tubular. The point of cardiac impulse is about the mammillary line at the 5th space 10 cm. from the median line.

February 16. The right chest is now flat to the 2nd rib. There is Skodaic resonance at the extreme apex. Respiration is distant. The point of cardiac maximum impulse is 11 cm. from the median line.

February 19. The patient has suffered a great deal from dyspnœa. He is aspirated and 240 cc. of fluid obtained.

February 21. Dr. Osler: The patient is resting quietly. He passed a comfortable night. Expectoration is not so foul. There is flatness almost up to the clavicle; Skodaic resonance on the clavicle. The breath sounds are distant; scarcely audible in the infraclavicular space. Tactile fremitus is well felt in the upper part of the chest and behind. There is absolute flatness in the back. The tactile fremitus is felt almost to the scar of the incision. There is dulness from the apex to the base; there is impaired expansion. The heart beat is below and within the nipple line. Extremely feeble breath sounds are heard on the right side.

February 28. Dr. Osler: There is Skodaic resonance in front to the nipple; distant tubular breathing.

March 2. The patient is lying on his back, the obliteration of the intercostal spaces on the right side is marked. The cardiac impulse is in the 5th space 10½ cm. from the mid-line. The resonance at the right apex is extremely tympanitic. In the erect posture there is flatness at the 2nd space and in the back to the spine of the scapula. Vocal fremitus is absent. Respirations are feeble and distant over the right side. The voice sounds are increased. There is no amphoric respiration. At the extreme apex the coin sounds are fairly well obtained. The succussion splash is perfectly clear. The flatness begins at the 5th rib in the mammillary line.

March 3. This morning there is slight suggestion of an amphoric character to the breath sounds. There is no metallic tinkling to be heard. In the erect posture flatness reaches to the clavicle. The succussion splash is, well obtained. Sputum yesterday was 180 cc. in volume. It is diminishing much of late. The coin sound is fairly well heard at the apex. The patient was transferred to the surgical side March 4, 1896.

Surg. No. 5199. Died March 4, 1896.

Operation, *paracentesis, tracheotomy.* The patient died on the table from obstruction to respiration from the pleural fluid in the bronchi. His condition was very bad. As soon as on the table he was seized with violent paroxysms of coughing and the rib was at once resected and a great deal of purulent, stinking fluid evacuated. His condition became moribund and tracheotomy was performed as a last resort.

Autopsy No. 781.

Anatomical Diagnosis.—*Chronic bronchiectatic cavity. Chronic pleurisy. Operation for empyema and pyopneumothorax. Acute œdema, left*

lung. Compression of right lung. Indurative mediastinitis. Hydronephrosis, kidney and ureter. Chronic orchitis. Tracheotomy wound, the edges of which are covered with dried recent hæmorrhages.

Body 164 cm. long, well nourished, strongly built. Rigor mortis in both extremities. Pupils medium size and equal.

Diaphragm on right side reaches 5th space, on left side lower border of 5th rib.

There is an oblique linear incision on right side which extends externally 12 cm. along chest wall. This incision is closed with silk sutures. There is a defect in the wall of the thorax owing to the removal of 4 cm. of the 6th rib, which leads directly into the pleural cavity and corresponds with the skin incision. On the posterior surface of the right thoracic cavity there is an oblique scar 10 cm. long, along the line of the 9th rib, which is healed, and corresponding to which there is an elevated scar-like mass of tissue about 5 mm. in height present in the parietal pleura on this side.

Left lung is free from adhesions and is voluminous. The pleura is dark in color and not thickened. On section a free escape of frothy serum both from the surface and from the bronchi, takes place. Consistence of lung is remarkably firm for an œdematous lung and cut surface is congested. The main bronchi are much congested and contain a thick, grain-like material upon the mucous membrane.

Right lung is compressed and along its posterior border it is adherent by extremely strong adhesions to the chest wall. The extent of the adhesions, which begin at posterior margin of lung, corresponds with 7 cm. of the width of the lung substance and 9 cm. at the base. At the base of the lung the adhesions to the diaphragm are so strong that they cannot be broken down and measure together with diaphragm itself not less than 2.5 cm. Along the anterior edge of the lung there are imperfect adhesions, which agree approximately with middle lobe, but over apex and mid-half of upper lobe exist firm adhesions to chest wall. The lower lobe is bound along its anterior margin and at the base. A cord-like strand of tissue extends from lower edge of lower lobe to the thickened diaphragmatic pleura. The pleural cavity itself contains only a small amount of thick grumous material, but upon the thickened pleura there is a collection of sticky, fibrinous pus which can be readily scraped off. The thickness of the visceral pleura, where it is free from adhesions is 2 mm. and of parietal pleura 4 mm. The slit in parietal pleura caused by operation measures 4.5 cm. The compressed portion of lung is dark and slaty, in lower part devoid, and in upper part containing a small amount of air. Occupying the entire extent of the posterior border of the lung, from bifurcation of the main bronchus to the base, is a large irregular cavity with gangrenous walls, into the side of which bronchus of the main bronchi directly enters. *These bronchi are ulcerated through a short distance beyond their origin.* The main ones which are affected would appear to be the side branches going to lower and middle lobes,

which, owing to adhesions, forked over into a single cavity. The branches which extend between lower lobe are dilated and their walls are weakened, but they are not ulcerated. The contents of the bronchi and cavity are grumous, semi-solid and of granular material, and give off a not very offensive odor. The capacity of the cavity could not be definitely estimated; it was longitudinal and would probably have held 500 cc.

In this rare and interesting case we may note the following points: The cause was evidently pneumothorax, due to the rupture of a very large bronchiectatic cavity. This was the diagnosis made by Dr. Osler. The operation of course removed the evidence which could have been furnished at the autopsy. After over three years of pulmonary disease with the formation of an enormous bronchiectatic cavity, in February, 1896, this by its rupture caused pneumothorax, as shown by the dyspnœa, the succussion splash, the dislocation of the heart, the distension of the side, the depression of the liver. The tympanitic resonance, absence of fremitus, and the coin sound, all present at the apex, show the condition to be pneumothorax, for the bronchiectatic cavity was present at the base of the lung, hence that cavity was not the source of pseudo-pneumothorax signs.

CASE 24.—*Pneumothorax of the right side, due to bronchiectasis.*
Steven K. Autopsy No. 544.

ANATOMICAL DIAGNOSIS.—*Tuberculosis of spine. Angular kyphosis. Extra-dural exudate. Compression of spinal cord. Tuberculous abscess of back. Lung tuberculosis. Tuberculous bronchiectasis. Perforation of pleura. Pneumothorax. Chronic tuberculous pleurisy. Tubercles in liver. Tuberculous ulcers in intestines. Calcification of mesenteric glands.*

Body much emaciated, surface pale, no œdema. Over the lower dorsal region of the vertebral column is a defect and there is a distinct prominence. On the left side of this eminence is a fluctuating tumor, the size of an orange, which on incision evacuates about 100 cc. of faintly red fluid, having distinct odor of iodoform and containing granular yellow precipitate. The walls of the cavity are covered with elevated granulations, to which adheres necrotic material of yellow granular precipitate. The entire wall is thick and dense and on section beneath the granulation is white and sclerotic. This cavity descends downward to the crest of the ilium, and the periosteum is thickened. In the depth the abscess extends to the vertebral column and is continuous with caseous material occupying the muscles connected with the column at the level of the 1st to 3rd lumbar vertebræ. The body of the second lumbar vertebra is excavated and the cavity is filled with caseous masses. About one-half of the body is deficient and there is caseous material in the second intervertebral disc, which is directly continuous with the contents of the cavity. There is an angular

deformity at this point which corresponds to lower end of the spinal cord, about 5 cm. above the filum terminale. At this point the spinal canal communicates with the defect of the body of the vertebra and there is an extra-dural, caseous accumulation compressing the spinal cord, which is thicker above and below at this point.

Left lung is bound by firm adhesions over the posterior border of its middle and over apex. The pleura is much thickened. The anterior edge of the lung is somewhat emphysematous. The inferior angle of the lung is adherent to the diaphragm. The pericardium is bound firmly to the lung. Beneath the surface are numerous elevated nodules. On section the lung is congested and there are miliary and larger tubercles. The lung is moderately pigmented and in both upper and lower lobes are a number of cavities with smooth walls, to many of which caseous material adheres and which communicate with bronchi. The largest one of this lung occupies the lower lobe posteriorly, is cylindrical in form, the bronchus is much atrophied and lung tissue about it is dense, slate-colored and sclerotic.

Right lung is firmly bound by old adhesions over apex and the upper portion of upper lobe; the lower lobe is retracted. The pleural cavity contains air but no fluid. There is a perforation the size of a hemp seed beneath the adhesions over the upper lobe, which communicates with a bronchiectatic cavity in the upper lobe. The upper lobe is retracted, pigmented and indurated and converted into a number of irregular cavities, for the most part of globular shape, which communicate with bronchi. In the lower lobe of this lung are similar cavities, but fewer. Caseous material and pus adhere to their walls. Other parts of the lung are congested, contain miliary tubercles, and larger tuberculous masses. Bronchi contain caseous pus. On section the lung substance is mottled dark red and black, and seems to be air-containing throughout.

Unfortunately the surgical history of this case was not to be found. The case was evidently due to bronchiectasis. It is to be noted that the perforation was just below adhesions.

Abscess of the Lung.—This cause of pneumothorax is excessively rare. If the descriptions given in Hippocrates be taken literally, and Littré considers we must, it was a more common cause then; but Adams thinks the same description may hold for a rupture of a tuberculous cavity. In 1841 Saussier [75] stated that there was but one case of this form on record. In 1855 Vallin [113] reported a case which he thinks belongs to this group. Gerard [127a] mentions this as a possible cause, but states that no case due to common metastatic abscess of the lung was on record. This is to us particularly interesting since one of our cases was certainly due to this latter cause.

CASE 25.—*Pneumothorax of the right side from perforation of a pulmonary abscess; empyema of the left side (primary in point of time); abscess of scalp; parotitis; death; autopsy.*

Fergus A. G., aged 26, white. **Surg. No. 12,734.**

Admitted November 29, 1901.

Died January 14, 1902.

Surgical Diagnosis.—*Empyema, necrosis of the skull, pleurisy, pneumonia, abscess of the lung, pneumothorax and parotitis.*

He complains of empyema.

Family History.—One sister died of consumption; otherwise negative.

Past History.—He has been perfectly healthy before the present trouble. He had the diseases of childhood, also probably typhoid fever. He has never been subject to coughs or colds. His habits have been very good.

Present Illness.—Began last June after a Turkish bath. The next morning he began to have pain in the left side but no chill. Temperature rose from 100 to 102. There was some cough but no expectoration. He was in a hospital for seven weeks. The diagnosis made was pleurisy. Two weeks before onset he had been aspirated and a clear amber fluid withdrawn, not more than half glassful. Then went to a health resort but grew very much worse. He thought he was going to die and was given up as a hopeless consumptive. The cough was incessant. He had no chills; diarrhœa was extreme; had frequent night sweats; he grew very weak and emaciated. The patient returned to the former hospital September 17. He was operated on immediately and thinks that a quart and a half of pus were evacuated from the left side. A rubber tube and gauze were put in. The sinus had almost stopped discharging by October 28 and then increased after the catheter was removed. He did not change much until two weeks ago when a physician injected a solution to wash out the cavity. The fluid did not return but the patient had a severe chill and was quite ill after this. The sinus has been discharging quite freely. The pus is of a reddish-yellow color and thick. The patient has gained 30 pounds since September 17.

Physical Examination.—November 29. The movement on the left side of the thorax is present but is less than on the right; especially in the lower axillary region is this discrepancy seen. Fremitus is present on the left side above the nipple but not below this point. Fremitus is not present in the left back below the angle of the scapula. On the left side, as far as the 5th interspace in the anterior axillary line, the note is about normal. Below this and about the wound, which is on the 7th rib, the note is impaired. Below the 7th rib posteriorly is an area about 6 cm. in diameter, dull on percussion. The breath sounds can be heard to the 7th rib on the left side but none below the sinus.

Backs.—About 4 cm. below the angle of the scapula on the left side fremitus is absent. The breath sounds are distant. The percussion note is dull. A probe can be passed into the sinus but does not enter any

definite cavity. The patient's general condition seems excellent. Since entering the hospital several ounces of thick, bloody pus have discharged. The heart sounds are clear. The relative dulness is not increased. There is no cough. The abdomen is negative.

December 2, 1901. Operation, Dr. Finney. Thoracotomy for an old left-sided empyema, resection of the 7th and 8th ribs, the lung partially expanded. The visceral pleura was not thickened. The diaphragmatic pleura was thickened. A drainage tube was sutured in. It was found that the rib, although divided at the previous operation, had not been excised. The abscess cavity was evidently located above and behind and a considerable quantity of pus was evacuated. The lung could be distinctly felt at the upper part of the cavity. There seems good prospects of its expanding.

December 4. The patient is uncomfortable and complains of the back of the head where there is distinct swelling and acute tenderness.

On December 7 this abscess was incised and a large abscess cavity found with the bone as the base, and with several smaller sinues leading off beneath the deep fascia of the scalp. Considerable pus evacuated. This abscess was over the left occipito-parietal region.

December 12. The scalp abscess is still discharging pus. The wound in the chest is draining well. The temperature is normal, the patient's general condition improving.

December 19. The patient has had a slight rise in temperature for the past two days, reaching 101 degrees. There has been a great deal of pain and hyperæsthesia in the back of the head and the whole of the scalp, especially in the left eye and the teeth of the left side. The pain was neuralgic in character, very severe. The eye grounds are negative.

December 20, 1901. Operation, Dr. Finney. Exploration of the skull. Excision of necrosed bone, drainage of an extra-pleural abscess; partial closure of the scalp with drainage. An area about 8 cm. in diameter of the skull was exposed. The bone on this area was roughened and denuded, corresponding to the suture between the parietal and occipital bones of the left side. Just along this suture there is evidence of extension of the process through the suture into the cranial cavity. A portion of bone was thus removed and an abscess cavity was found outside of the dura connected with the necrotic bone. There was no evidence of extension through the dura. The abscess cavity contained about one and a half drachms of thick, yellowish pus.

December 25. During December 22 and 23 the temperature was nearly normal. Yesterday the patient complained of chilly sensations and slight nausea. The temperature rose to 104.4. This morning it is still above 103. There is no cough; an occasional pain in the head. The urine is negative. The heart is negative. The lungs are negative. Both wounds appear in good condition. Leucocytes 17,600.

December 28. The patient's temperature has continued elevated. To-day for the first time the patient developed a little cough and complains of pain down his sternum when he takes a deep breath. Examination of

the lungs shows the same area of dulness and absence of breath sounds at the left base noted before his first operation. The right side is negative. Just over the 2nd and 3rd costal cartilages is heard a faint friction rub, synchronous with the heart beat. Just inside of this area are heard a few moist râles. There is no increase in the area of cardiac dulness.

January 5, 1902. Dr. Thayer: The cardiac maximum impulse is visible in the 5th space about 10 cm. from the mid-line, just in the mammillary line. Both upper fronts move with inspiration, the left perhaps a little more than the right. The resonance is clear above the incision on the left. On the right, as one passes down the note over the 4th rib becomes rather tympanitic. Flatness at the upper border of the 6th rib in the mammillary line. In the axilla there is a dull tympany reaching anteriorly to within about 4 cm. of the mammillary line. Flatness at the 6th rib in the mid-axilla. The vocal fremitus is felt throughout the right side. Respirations loud and clear in the left front until one approaches the cardiac area, where a faint, superficial, dry crepitus is heard in the 2nd and 3rd interspaces, chiefly on inspiration, slightly on expiration. It sounds pleural and does not seem to be increased by coughing. On the right side respiration is also clear but rather harsh above. In the 2nd and 3rd interspaces are fine crepitations on inspiration, not apparently affected by coughing. In the axilla the respiration is enfeebled but no adventitious sounds are to be heard. On the anterior border of the dull area is a little dry crepitation. In the posterior axilla the respiration is almost absent and the voice sounds reach the ear entirely from the outside.

Right back.—There is flatness below a line beginning about the spine of the scapula. The left back is clear. Fremitus is nearly absent over the flat area. In the supraspinous fossa respiration is feeble, an occasional crackle is heard with inspiration. Below, the sounds have a distant tubular character, accompanied by dry crepitation. The voice sounds are well heard. Occasionally there is a nasal quality. At the left base there are also numerous crepitant râles, especially with inspiration. At the extreme base, respiration is a little blowing in character, though everywhere louder than on the right side. In the upper half of the left back the breath sounds are clear. Heart sounds are clear excepting for a rather scratchy sound heard in the pulmonic area with systole.

January 8. An aspirating needle was inserted in the 8th interspace of the right back just below the angle of the scapula and 240 cc. of slightly turbid, reddish-yellow fluid were withdrawn. Microscopically, no organisms were found. No tubercle bacilli were found.

January 10. The patient developed to-day a parotitis on the right side.

January 13. The patient has been gradually getting worse. Respirations are much more labored and rapid to-day. When seen this evening he was in great dyspnœa, and this afternoon the coin sound is very well obtained on the right side.

Dr. McCrae: The amount of movement of the right thorax is very striking. It moves as a whole and the shoulders and the upper arm are

lifted. The costal margin moves almost an inch below the skin. The patient is in great dyspnœa and distress. The striking thing is the great movement on the right side. This is apparently due to the use of all accessory muscles and while at first glance it suggested marked expansion, on closer examination it was seen that practically there was no expansion on the right side. The patient's previous trouble has been on the left side. The onset has been very sudden. On percussion the note is markedly hyperresonant and almost tympanitic on the right side. The tympany comes down unusually low, reaching to the costal margin in the parasternal line, in the mammillary line to the 8th rib. In the mid-axilla to the 9th interspace. On auscultation over the upper front there is practically nothing heard. Below the 4th rib are rather feeble breath sounds which below the 6th rib are definitely amphoric. At times some dry and liquid râles are heard which at times have a tinkling, metallic quality. Over the greater part of the back there is impairment but in the 9th and 10th interspaces of the scapular line is a most intense amphoric percussion note and the breath sounds have an intense amphoric quality. The coin sound is not constant. The condition is probably a localized pneumothorax due probably to the conditions in the lung itself. The cardiac impulse is in the 5th interspace just in the mammillary line.

January 14. The patient died at five o'clock to-day. He had been gradually getting weaker and more dyspnœic.

The urine while in the hospital was negative; no albumin, no casts. The fluid obtained at the operation of December 4 gave cultures of staphylococcus pyogenes aureus. The same organism was obtained from the pus of the extra-dural abscess.

The temperature on admission was normal. After the operation of November 2 it rose and for three days ranged from 100 to 102. It then fell slowly to normal which it reached December 11, and there remained until December 15, when it began to rise, reaching 103.6 on December 20. It then fell, reaching 99 on December 23, but on the following day rose to 104.4. It remained elevated from 101 to 104.6 until death.

Autopsy No. 1848.

Anatomical Diagnosis.—*Pulmonary abscesses at bases of both lungs. Perforation into right pleural cavity. Pyopneumothorax. Chronic pleurisy of the left side. Sinuses to base of lung. Scalp wound with removal of underlying bone and exposure of dura. Chronic appendicitis.*

Body is that of an emaciated man, 170 cm. in length. Upon occipital region of the head, to the left of the median line, is a wound about 3 cm. across, forming the angle of junction of two incisions at right angle to one another, each about 7 cm. in length. The skin is raised and beneath, it is packed with a small amount of gauze. The dura mater is exposed, the bone having been removed. The wound is clean and dry and shows beginning granulation. The abdomen is distended, particularly at and to the left of the umbilicus.

The right pleural cavity, tested before opening, is found to contain air which bubbles out under water apparently under considerable pressure. The lung is compressed to the posterior and inner part of cavity, and the latter contains 250 cc. of creamy white, thick, purulent fluid in which there are a few semi-solid particles of the same white color. Upon the outer surface of the lower lobe, near the base of the posterior border, is a perforation of the lung from which oozes purulent thick fluid, similar to that within the cavity. The posterior border and inner surface of the lung is bound to the chest wall. The left pleural cavity contains no excess of fluid. The lung is very densely bound to the diaphragm and its outer surface.

Right lung is of small size and collapsed. The pleura over the surface of both the upper and lower lobes has a dull yellow-grey color and is covered in places with a layer of tough yellow fibrin, mostly about a perforation, which is situated 3 cm. from the diaphragm. This perforation has fairly sharp edges, is round and 4 mm. in diameter. The cavity containing pus is walled off from the remainder of the pleural cavity so that the anterior aspect of the lung's surface is smooth and separated by a line of fibrinous adhesions. The lung substance is tough and flaccid, does not crepitate and is of an almost homogeneous dull red color and contains fairly abundant fluid. Although it is not absolutely consolidated, a bit cut out sinks in water. The abscess cavity is situated immediately below the outer surface of the lower lobe, it is irregular in shape and approximately 3 cm. in diameter, sharply marked off from the surrounding substance by a membrane about 0.5 mm. in thickness, composed of tough, yellowish-grey material. A vessel passing through the cavity is lined by a similar material. The cavity contains soft pus, like that in the pleura, and at the same time a mass of semi-solid, opaque yellowish material. The lung tissue about appears compressed but is not consolidated. The lower margin of the cavity is about 1 cm. from the diaphragm and the tissue about contains a few rather soft yellow nodules, somewhat granular on section, the largest about 2 mm. across, having the appearance of a minute area of bronchopneumonia. No large bronchus could be traced to the cavity, but a small bronchus, not more than 2 mm. across, with soft membranous walls, enters the lower aspect of the cavity. All of the bronchi to the lower part of the lung contain thick, mucopurulent material, similar to that occupying the abscess cavity, and their walls are moderately red and injected.

Left lung contains air and is crepitant except at the lowermost part of the posterior border of the lower lobe. Here the tissue is firm in consistence and of a deep red color, but quite flaccid. On section into this area a small abscess cavity, 1.5 cm. across, is found immediately below the pleura. Its contents are similar to those of the cavity found in the right lung, and in the tissue about are a few smaller abscesses. The bronchi are injected, particularly those of the lower lobe, and contain mucopurulent fluid. The pleura over the posterior and outer surface of

the lower lobe is firmly adherent to the chest wall, through which in the axillary region, there is an opening from the skin surface communicating with a small sinus which ends blindly in contact with the visceral pleura, which is thickened and granular.

This case shows an interesting sequence of left-sided pleurisy with hydrothorax, evidently poor operative treatment, hence subsequent pyothorax on the left side. On admission to this hospital the empyema on the left side was operated on December 2, 1901. Two days later a scalp abscess was incised and two weeks still later the skull explored. December 28 a right-sided pleurisy developed and the symptoms from an abscess of the right lower lobe which on January 13 ruptured, causing pyopneumothorax. On January 10 a parotitis had developed.

The signs of pneumothorax were, the violent attempts at respiration, the hyperresonance on percussion over the front and especially at the base behind. Over the front the breath sounds were almost absent, but at the base behind, hence just opposite the perforation, is intense amphoric breathing.

This case is of particular interest since it is one of very few, or indeed the only one, on record due to a metastatic abscess.

The following may belong to the same series:

CASE 26.—*Pneumothorax of the right side; due to metastatic pulmonary abscess.*

Annie Z. Autopsy No. 856.

ANATOMICAL DIAGNOSIS.—*Puerperal infection; perforation of vagina and bladder. Infectious thrombosis of pelvic veins, inferior vena cava and renal veins. Metastatic lung abscesses. Acute fibrinous pleurisy and pneumothorax. Large white (puerperal) kidneys. Acute splenic tumor. Probably post-mortem invasion of gas-forming bacilli.*

Body of a strongly built, well-nourished woman, 163 cm. long. No subcutaneous œdema. Surface of yellowish hue. Pupils middle width and equal. Abdomen enormously distended. Slight post-mortem discoloration. Posterior surface purplish discoloration. Neck distended; tissues crackle upon pressure. Crackling in inguinal region.

Diaphragm on the right side at costal margin; fluctuating; on incision under water much gas escapes. On left side diaphragm is at 4th rib.

Left lung is voluminous; lower lobe much congested. Bronchi are blood-stained, vessels free.

Right lung compressed and airless. Covering the pleura over the lower lobe is a thick layer of firm, yellow fibrin which causes adhesions between this lobe and the diaphragm. Bronchi and vessels are free. There is no excess of fluid in this cavity. On section of this lung two

abscesses, filled with thick yellow, creamy pus, are discovered beneath the pleura of the lower lobe. No perforation of the pleura discovered.

Most unfortunately the clinical history of this case is not to be found.

At autopsy gas was present in the right pleural cavity. Certainly pneumothorax was present, certainly metastatic abscess of the lung. From the heart's blood the Bacillus aerogenes capsulatus grew but not from the contents of this pleural cavity from which grew the Streptococcus pyogenes and the Staphylococcus pyogenes aureus.

Gangrene of the Lung.—Laennec [41] considered this as one of the causes of pneumothorax, the gas arising from decomposition of the gangrenous tissues. Saussier [78] divides such cases into those (1) in which the gas arises from decomposition of the gangrenous tissue, in which case it will be fœtid, and (2) those in which the gangrenous process merely perforates the lung. He expressed himself as surprised that this disease does not oftener cause perforation of the lung, since contrary to tuberculosis the pleura is widely destroyed together with the tissue of the lung and there is no possibility of the formation of adhesions. The cases of pneumothorax due to pulmonary infarction, at first considered a separate cause, probably all come under this head, the infarcted area becoming gangrenous. Among the cases reported we may mention Benda's cases,[95] Rumpf,[96] in which the sequence was pneumonia, bronchiectasis, gangrene; Monneret,[104] who reported a case of gangrene following pneumonia; Weil [201] reported two cases with recovery. Ewart [259] reviewing the literature on this point reports an interesting case with recovery. This youth, 18 years old, presented the sequence of influenza, bronchopneumonia, emphysema, gangrene, operation and recovery. Comby [293] adds one more case. Zuppinger [358] states that this cause is more common in children than in adults and reports three cases, in one of which the sequence seems to have been diphtheria, thrombosis of the pulmonary artery, infarction, gangrene. Gerard [127a] also reports a case usually counted among those due to pulmonary apoplexy, and Lyon [161] one due to embolism of the artery, the embolus originating in the femoral vein. We add one case especially interesting, since it was a complication of typhoid septicæmia:

CASE 27.—*Pyopneumothorax of the right side; due to gangrene of the lung, in a case of typhoid septicæmia with thrombosis of the pulmonary artery.*

(This case was reported by Drs. Flexner and Harris, *Johns Hopkins Hospital Bulletin*, Dec., 1897.)

W. G., male, 68 yrs., white. **Med. No. 7969.**

Admitted Oct. 28, 1897.

Died Oct. 30, 1897.

Complained of shortness of breath.

Past History.—Unimportant.

Present Illness.—This began two months ago, since which time he has been losing weight and strength. Appetite poor; no fever, chills or night sweats. Two weeks ago he suffered from severe pain in the back, followed by shortness of breath and abdominal pain.

Oct. 26. While undressing he fell to the floor. He was very quiet the next day, not moving from the position on which he had been placed on the bed.

Physical Examination.—The patient is very dull and listless, seems to be in much pain, groaning with each expiration. Respirations are 44 to the minute. The thorax is somewhat barrel-shaped. The right side is more prominent than the left; the percussion note hyperresonant in front and back. Respiration is harsh, expiration is prolonged. There is a loud friction rub in the axilla.

Heart.—Point of maximum impulse is in the 5th space 10 cm. from the mid-line. The absolute dulness begins at the 4th rib at the sternal border. The spleen was not palpable. Examination of blood negative for malarial organisms, leucocytes 18,000. The patient gradually sank and died, Oct. 30.

Autopsy No. 1013.

Autopsy one hour after death.

Anatomical Diagnosis.—*Typhoid fever without intestinal lesions. Typhoid septicæmia. Thrombosis of main branch of pulmonary artery supplying lower lobe of right lung. Gangrene; perforation of pleura; pyopneumothorax. Acute fibrinous pleurisy. Acute splenic tumor. Parenchymatous degeneration of kidneys. Obliteration of appendix vermiformis.*

Body of a large, strongly-built and moderately well-nourished man. The surface of body presents a sallow hue. No subcutaneous œdema. Rigor-mortis present. Slight patchy livor-mortis on posterior aspect of trunk. The right half of chest is more prominent than the left; on percussion a highly resonant note is elicited. Thorax on this side resonant to within a few cm. of costal margins.

Diaphragm on right side 7th rib. On left side 6th space. The diaphragm gives a sense of being pushed away down on the right side by air. Opened under water and gas escaped freely.

On removing the sternum and costal cartilages the right lung is found compressed. The upper lobe is pressed against the back of the thoracic cavity and vertebral column. It is quite airless. The middle lobe is not distinguishable, the lower lobe is more voluminous and is placed

closely against the diaphragm. The upper lobe is free from adhesions except along its upper border near the apex where are old fibrous ones found to exist. The lower lobe is bound firmly to the diaphragm by old adhesions. The pleura covering the sternum on the right side over the thorax generally is thickened; it is also covered by a shaggy, fibrinous layer. But a much thicker layer of fibrin, quite shaggy in character and containing much serous fluid covers the anterior and lateral surfaces of the upper lobe. On removal from the thorax the lower lobe is voluminous, dark in color and consolidated, but not very firmly. The pleura is covered, except where the lung is bound to the diaphragm, by fibrinous pseudomembrane, also dark in color. On removing this several defects make their appearance in the visceral pleura over the anterior surface. These were quickly seen, but the whole pleura was necrotic, so that any violence easily brought about the production of others. A probe easily passed through these into the very friable lung substance. The bronchi are quite free. But the main branch of the pulmonary artery going to this lobe was occupied by a quite firm, partially decolorized thrombus. From it more recent, red, soft thrombi extended into the communicating vessels in several directions. The oldest appearing thrombus was the main mass; no older ones could be found. As many large branches as possible were cut up. On section the lung is dark greyish in color and has a moderately offensive odor. About 75 cc. of slightly blood-tinged fluid was present in the cavity, but considering the water test, this might have gained entrance through the abdominal cavity.

Left lung bound by old adhesions over the apex. A circumscribed area of thickening of the pleura existed here. In the substance of the lung, in the apex, a cretaceous mass the size of a marble existed. No consolidation elsewhere. Bronchi and blood-vessels free.

Spleen.—Free from adhesions. Weight, 160 grammes. Capsule tense; consistence diminished. The cut surface is dark in color.

Heart weighs 305 grammes. All the valves sufficient. The aortic leaflet of the mitral contains several opaque fatty patches. Aorta shows patches of thickening. Coronary arteries patent; show patches of thickening. Heart's flesh firm; dark in color.

Aorta generally shows nodular thickenings; no calcification.

Liver weighs 1670 grammes. Over the lower third of the right lobe, anterior surface, the capsule is opaque and thickened and at the upper margin is a well-marked tight lace groove. On section the surface of the liver is dark in color. No abnormality noticed. Adhesions between the omentum and gall bladder.

Kidneys are alike. Combined weigh 305 grammes. Capsule strips off easily. No adhesions; surface is smooth; a few small cysts in the substance. On section cortex is swollen; striæ coarse. Consistence not increased. Mucous membrane of pelvis quite normal. Ureters normal.

Urinary bladder normal. Veins at base contain soft, recent, dark thrombi.

Gall bladder moderately distended, bile thick and dark colored. A large number of concretions, very regular in size and pale in color, not exceeding a hemp seed each in size, in the bladder. Ducts normal.

Pancreas, adrenals, thyroid appear normal.

Stomach, œsophagus and intestine apparently normal except appendix. The appendix vermiformis is involved in adhesions, lies behind the cæcum and is transformed into a fibrous cord. Mesenteric glands not enlarged.

Dura mater firmly bound to the skull cap. The pia-arachnoid contains a small excess of clear fluid. The brain itself appears on section normal.

Cultures were made from the heart's blood, brain, both lungs, spleen, liver and kidneys. Those from the blood and brain were negative, but from all the other organs the bacillus typhosus grew. I will not give the long detailed bacteriological report but merely say that the organism was the bacillus typhosus culturally, and responded typically to the Widal agglutination test with the serum of a known typhoid patient.

This case is of particular clinical interest as we know of no similar one on record. The cause of the pneumothorax was gangrene of the entire lower lobe of the right lung due to thrombosis of the pulmonary artery. The patient died of a septicæmia of the typhoid bacillus. There were no intestinal lesions. On the autopsy table the right side of the chest was distended and hyperresonant, the diaphragm pushed down. Gas escaped by the water test. The pleura of the lower lobe was entirely necrotic. Several defects in it were found. The odor was offensive.

Rupture of the Lung.—*Cases Due to Severe Injury.*—That rupture of the lung occurs frequently is a well recognized fact, but since the pleura does not at the same time rupture, the condition of interstitial emphysema results. The air may dissect its way through the interlobular tissue to the mediastinum and thence to the neck, from which point it will spread over the body. Such cases are not rare. The whole subject receives very careful consideration by Demarquay.[129] The best illustrations are those due to run-over accidents without fracture of the ribs. These cases Demarquay considers occur more particularly in the young, since it is necessary that the thorax should be very flexible. The mechanism usually given is that the glottis is closed, the chest immobile. The pressure occludes a bronchus, and then the pressure passing towards the periphery causes the imprisoned air to rupture the lung. Many such cases are on record. Among these are Curling's,[135] in which case the boy was run over by a cab; Butlin;[150] Gould.[153] This

last case is particularly interesting, since the pneumothorax developed two days later. In Lucas's [256] case the child was two and a half years old. It is he who attempts their explanation. But these cases are not all of children. Marsh's [152] case was a man who had been run over and who presented the extraordinary condition of the lung torn away from the bronchus. In none of these cases were ribs broken. Other similar accidents are illustrated by the cases of Jaccoud,[204] whose patient was a man crushed between two wagons; Butlin,[150] whose patient was a man caught between the bumpers of two cars and who presented the symptoms and signs of pneumothorax but autopsy showed a rupture of the diaphragm and the presence of the stomach and transverse colon in the pleural cavity; Butlin,[150] whose patient developed pneumothorax as the result of a severe fall; Ross,[168] whose patient was a healthy man struck in the back by the pole of an omnibus; Schuh (Skoda [110]), whose patient was crushed between two cars and recovered. But even severe crushes of the chest do not often result in pneumothorax, as Biach found, who collected 227 cases from the hospitals of Vienna and not one of them with pneumothorax.

Cases Due to Less Severe Injury.—Among these may be mentioned the cases of Abeille,[134] whose patient, a man in excellent health, woke up one night with pneumothorax. He gives the history of having strained himself somewhat the night before. Delgrange's [167] patient was a coppersmith who was showing the strength of his lungs; Johnson's,[182] was a boy 15 years old who ran twenty miles and three days later developed pneumothorax; Adams' [202] case was a man who developed pneumothorax while straining at stool. This case is particularly interesting, since the signs were present for four years but without any effusion during the time. Gibson's [273] patient developed his as a result of dumb-bell exercise; Chauffard's [274] patient was a woman who picked up her child with her right arm at the same time she was laughing and developed pneumothorax. This case is of particular interest, since it is the first on record in which tuberculin was used to show the non-tuberculous nature of the lesion. Kidd's [286] patient fell from an omnibus. He received no fracture of the ribs but pneumothorax followed with effusion and slight fever; Morris's [355] patient had fallen twenty feet; Levison's [314] patient was a healthy man who had some shortness of breath while climbing a mountain, and later while laughing developed pneumothorax.

"Spontaneous Pneumothorax."—Cases without History of Injury.—An ever increasing number of cases are now being reported. In some cases careful questioning has shown that a slight strain, but not an unusual one, had preceded. Since such slight strains are of course common, we will include such cases in this list. Among the cases on record are those of Flint,[115] pure pneumothorax developing in a peddler; Thornburn,[120] whose patient developed pneumothorax at night after a day in which he had been taking unusual exercise at rowing; this is quoted as the first spontaneous case of pneumothorax on record. In Biermer's [121] it developed in a boy as the result of dancing; in Noble's [122] case it followed unusual exercise and developed at the breakfast table. He also reports another case,[144] in which the man awoke one night with pneumothorax. In a case of Vogel [131] the pneumothorax developed like a flash of lightning from a clear sky. In Wilks' patient [148] it developed while doing his ordinary work as a bill sticker; Church,[151] Waller,[231] Weber,[332] Galliard,[343] Jochmann,[351] also report cases. Hall [207] collected in all 31 cases of pneumothorax in apparently healthy persons. He reported five. He considers that a strain always precedes. West [209] says that such cases represent from 2 to 3 per cent of the total cases of pneumothorax but emphasizes the fact that the lungs are not normal and cites experiments on this point. Galliard [218] gathers 22 cases. He discusses their cause and denies that fever and succussion are ever present. According to him it develops particularly in men between 17 and 37 years old and is always total. He considers the rupture of an emphysematous bleb to be the common cause. In Pitt's case [383] there was no previous examination. The patient died of hæmopneumothorax and at autopsy an emphysematous bleb was found which had been torn by a pleural adhesion. Fussell and Riesman [350] report 2 cases and collect 56. They say that it occurs particularly in young men and is rare. They found that pleurisy was present in the chest but in one case and that rarely is there any fever. According to them all cases recover. Maillart and Lasserre [357] report a case developing in a man stooping over. They collect 51 other cases, 16 of whom were between 14 and 20 years of age; 45 were men; 28 of 50 of these cases gave no history of a previous strain; 10 per cent were fatal, and what is very interesting, 7 of the total number had recurrences.

Belonging in this same group may be mentioned the following cases in which the immediate cause was nothing extraordinary.

Laughing.—Benjamin Bell [34] mentions this as a possibility. This was considered the cause in Levison's [314] case. M'Dowel's [114] patient while laughing felt a rupture of the lung.

Yawning.—Boland [329] attributes the development of pneumothorax in his case, a healthy person, to this cause. The man, however, woke up with a pneumothorax soon after retiring. This case is particularly interesting since it is one of the few cases of idiopathic hæmopneumothorax.

Crying.—Again Benjamin Bell considers this as a possible cause. The huckster which Klemperer reports [231] owed his pneumothorax to the effort of calling his wares. There was no suspicion of tuberculosis in this case.

Interesting illustrations will be found under the heading, "Pneumothorax in Children."

Sneezing.—The only case found is that of Stiller.[338]

Coughing.—Benjamin Bell [34] considers this a possibility; Widal [216] mentions a case of Ramskill; Murdoch [244] considered his case due to this alone.

In this connection would of course occur many cases developing in tuberculosis, diphtheria and whooping-cough which will be found under those headings.

Emphysema of the Lung.—The importance of emphysema as a cause of pneumothorax has grown of increased importance. Laennec [41] was not sure he had seen such a case. Devilliers,[50] seven years later, wrote a thesis on this subject, reporting one very clear case and discussed its symptomatology and diagnosis. Saussier [78] considered this the best proven variety and also mentioned the interstitial emphysema as the cause of the so-called subpleural pneumothorax. Delosea [103] reported a case with death in three hours and discussed the question of essential pneumothorax. The cases reported by Hughes [107] are interesting, in one case the emphysematous bullæ varying in size from a pigeon's egg to a pea. In 1887 Augry [208] collected all the cases he could find in literature. The best paper on this subject, however, is that of Zahn,[240] who classifies the cases in a very satisfactory manner. He considered it a rare cause, only six cases with autopsy appearing on record. These he divides into the following groups:

(1) The rupture of an emphysematous pulmonary vesicle.

(2) The rupture of a bleb of interstitial emphysema. This cause he says explains most of the cases of pneumothorax due to strain.

(3) A tear in the pleura due to an old adhesion. He reports one case and quotes one from literature.

(4) Atrophy of the pleura, of which he gives one illustration.

He considers its predominance in men to be explained by the greater frequency of chronic bronchitis in this sex.

Since the disease weakening the lungs in cases of spontaneous pneumothorax is hard to determine, the following cases are inserted here:

CASE 28.—*Spontaneous pneumothorax of right side.*

(This case was reported by Dr. Osler in the *Maryland Medical Journal*, 1897-8, Vol. 38, p. 461.)

Henry W., 38, white. **Med. No. 8233.**

Admitted January 20, 1898.

Discharged April 13, 1898.

DIAGNOSIS.—*Pneumothorax.*

COMPLAINS of shortness of the breath, heaviness in the stomach and pain in the shoulder when he stands up.

FAMILY HISTORY.—Very good. No history of tuberculosis.

PAST HISTORY.—Has always been very healthy. He had malaria when ten years old; has been treated at the dispensary for indigestion off and on for five years. Has had rheumatic pains in the arms for twenty years, but never stopped work on account of them. Has had no diseases. Habits have been good. Has not lost weight or strength. For 22 years has worked in an oyster house.

PRESENT ILLNESS.—On January 14, 1898, six days before admission, he was sitting quietly playing cards and eating his lunch in the oyster house, waiting for the bell which rings whenever a fresh supply of oysters arrived for shucking. He says he got up to raise a window, which he did without effort, but felt stabbing pain in the back of a rather unusual character. About twenty minutes later he started to go down the stairs to work. On the way down he was suddenly seized with a feeling of great pressure and suffocation, the pressure being chiefly in the epigastrium. He went to work, but in about two hours' time such severe pain of a stabbing nature between the shoulders began that he had to stop work and started for home, a distance of seven blocks. On the way he suffered greatly from suffocating feelings, pressure in the abdomen and pains between the shoulder blades. With difficulty he reached home and went immediately to bed. In a few hours he developed a cough, but did not expectorate for a day or so. The cough was paroxysmal, the expectoration scanty, of a bluish white color. The severity of the symptoms gradually diminished. He was in bed until the 18th, when he felt well enough to go out for a walk, but

after a short distance had a second attack similar to the first. He with difficulty reached home and went to bed and remained there until the time of admission to the hospital. Until this present illness the patient has felt well, has had no cough whatever. Now he says he feels perfectly well while sitting or lying down, but if he stands up or moves much he is confident he will bring on an attack. Has had no chills or chilly feelings; no vomiting; no nausea.

Physical Examination.—Patient is a well-nourished, healthy looking man. On inspiration there is very little difference in the appearance of the two sides of the chest. Cardiac impulse is seen in the 5th interspace below the nipple. The motion of the right chest is nearly absent below and is slight above. Vocal fremitus is much diminished throughout this side. On percussion the note is normal throughout the right front, resonance extending nearly to the costal margin in the recumbent position. No movable dulness. In the back the right side appears slightly fuller than the left. Note throughout is slightly higher pitched on the right. Respiration in the upper left front is somewhat harsh, puerile and wavy. On the right in the supraclavicular fossa there is very faint, distant, somewhat amphoric respiration. Throughout the front the breath sounds have the same distant amphoric character. At the right base this far-off metallic quality to the breath sounds is very marked; the coin sound is very distinct. In the left front there are no adventitious sound to be heard. No tubercle bacilli are found in the sputum.

Liver dulness begins at costal margin and extends 6 cm. below.

Blood count.—Reds, 6,208,000; whites, 5200.

January 25, 1898. There is very little fluid in the chest, the only sign of its presence being succussion. There is no metallic tinkle on coughing.

January 27. Dr. Osler: General condition is excellent, looks well, feels well, no fever. In the 6th interspace there is slight movable dulness but the note is clear to the base behind. Succussion can be heard faintly in the right axilla.

January 31. No signs of effusion now present.

February 2, 1898. Dr. Osler: The note on the right chest sounds like that of extreme emphysema, amphoric breathing most marked in the lower mammillary region, not so well heard in the supraclavicular region, especially on expiration. Apex beat has moved somewhat in. No metallic tinkling.

February 7, 1898. Tuberculin test was tried. No reaction followed (2 mg. used). This was repeated with 4 mg. on the 21st. Still without reaction.

February 23, 1898. Dr. Osler: The amphoric quality of the respiration has rather increased; no succussion splash. The patient was given Wolffe's bottles with which to exercise the lungs.

March 18. The patient has lost almost completely the amphoric quality of the breath sounds.

March 28. The right side moves more than the feeble inspiration would indicate. Coin sounds not well heard. Respiration practically absent over the greater part of the back and feeble in front. Metallic ring quite absent. Point of maximum impulse in the 5th interspace 10 cm. from the midsternal line.

April 13. Patient in splendid condition. The lung on the right side is completely expanded. Respiratory movements are equal and good. Percussion note slightly duller over the entire right front and back. Not hyperresonant as formerly. Auscultation shows a number of râles of rather leathery character, especially in the right scapular region. Not much influenced by coughing and mixed with a number of coarse, crackling râles. Breath sounds on this side are feeble but have no tubular modification. Point of maximum impulse in the 5th interspace 9½ cm. from the midsternal line. Liver dulness begins in the 6th interspace; lower border not palpable.

SUMMARY.—The patient was in the hospital two months and twenty-four days suffering from pneumothorax of the right side showing only slight changes of effusion for the first few days. After this time the pneumothorax remained pure. The tuberculin test was twice negative. He was discharged well to-day and has gained eleven pounds while in the hospital. The temperature remained normal during the whole of his stay.

This rare case deserves especial attention. The pneumothorax, of six days' duration, was very evident on admission. The onset with sudden stabbing pain between the shoulders, the feeling of pressure in the epigastrium and of suffocation are very suggestive. On admission the limited motion on the distended right side, the diminished fremitus with resonant percussion note throughout, the faint, distant amphoric respiration and the distinct coin sound leave no doubt as to the diagnosis. At first the pneumothorax was pure, but five days after admission the slight succussion splash showed some had formed.

On February 23, one month later, the succussion splash could not be obtained, the amphoric respiration sounds had rather increased, but now the right lung was certainly taking more part in respiration. Three months later he was pronounced well.

But what was the cause of this pneumothorax? This is one of the very few cases on record in which one can state positively that it was not latent tuberculosis, for the tuberculin test carefully used was negative. The manner of onset and its non-febrile course led Dr. Osler to place it among the emphysema cases. It is of interest that there was a slight pleurisy resulting, as shown by the slight

exudate and the pleural rub on dismission, and this may be an argument against the assertion so boldly made, yet not very well proven, that the presence of air in the pleural cavity is harmless. It is of interest also that at no time were metallic tinkles heard, also that the breath sounds were distant and amphoric and only became louder as the patient used the collapsed lung more.

The exertion causing it was not an unusual one.

CASE 29.—*Pneumothorax of the left side, due to overexertion* (?), *with very long* (?) *course.*

J. B. D., male, aged 40. **Med. No. 11,439.**

Admitted June 18, 1900.

Discharged June 23, 1900.

DIAGNOSIS.—*Locomotor ataxia; chronic pneumothorax.*

The patient complained of hæmorrhage from the bowels, difficulty in walking and shortness of breath.

FAMILY HISTORY is negative; no tuberculosis.

PAST HISTORY.—Has had some of the children's diseases—measles, whooping-cough, scarlet fever. When a young child was perfectly well so far as he knows. Denies typhoid fever and pneumonia. Was strong and healthy until present illness. Has always been an active man, but never did hard labor. He denies any venereal infection. He denies to the habitual use of alcohol. He has two children living and well; another died of "tubercular meningitis."

PRESENT ILLNESS.—About fifteen years ago the patient ran a one-hundred-yard race, at the end of which he had suddenly a severe pain in the left axilla. He was unable to run a second race, as he had intended to do. He was driven to the doctor's. The pain constantly became more severe, the shortness of breath increased. After this he remained in bed six months on account of the pain, weakness and shortness of breath. The doctors were undecided as to the nature of the trouble, one diagnosing "rupture of the air sac of the lung." The patient has never felt well since his injury, but has been able to do light work until six months ago. He has had during the whole time a chronic diarrhœa. About twelve years ago he had hæmorrhage from the bowels lasting two or three days, and thinks he lost a quart or more of blood. During the past twelve years he has had eight or nine similar attacks, almost one a year. At times he noticed small fresh clots between the larger hæmorrhages. The patient has noticed numbness in the feet and ankles one year. He was able to walk all the time, but did not notice any incoordination as at present. This lasted about one month. About two months ago the patient noticed difficulty in walking and stamping gait, and would fall if he shut his eyes. Five months ago he had shooting pains in the legs. Since then these have improved. During the past four or five months he has noticed a sensation as if a tight band were drawn about the lower part of the chest. At times

he has nausea, but no vomiting. For the past few months the patient has noticed a loss of memory, cannot think what he wishes to say. He has no cough, slight difficulty with vision. At times during the past seven years when the patient jumps or moves suddenly he has noticed a hollow ringing sound in his chest as in an empty bladder.

Physical Examination.—The patient is a well-nourished man; no respiratory distress. He walks with a stamping gait, shows marked signs of muscular incoordination of the lower extremities; Romberg's symptom is very well marked. It is with great difficulty that the patient can walk without a cane. He is not anæmic. The pupils react well to light and accommodation. The chest is well formed. The clavicles are equally prominent. There is very slight bulging in the lower left axilla; no retraction or obliteration of the interspaces on either side is to be made out. Respiratory movements are about equal on both sides. Vocal fremitus is very much diminished but present over the entire left side, especially in the lower left axilla. On auscultation the breath sounds over the entire right side are normal. Over the left upper front they are much suppressed but are well heard and of normal quality. In the lower left front and axilla the breath sounds are still more faint and distant. At the level of the 4th rib a few distinctly metallic tympanitic râles were heard but not after the first examination. On percussion the note is resonant over the entire right front. It is resonant but considerably impaired over the entire left front down to the upper border of the 5th rib in the mammillary line. The note becomes decidedly higher pitched and tympanitic in quality below this point. This area moves definitely when the patient sits up and changes position. Over the left back, down as low as the level of the 9th rib with the patient sitting up, the breath sounds are suppressed and distant. Below this line they are very distant. They have no amphoric quality. Vocal fremitus is faint over the upper left back, almost absent below. On percussion the note is somewhat impaired above, becoming higher pitched and tympanitic below. The coin sound is very well marked over the lower chest, especially below. With the patient sitting up it is not well heard above the 5th rib. There is no succussion splash obtained.

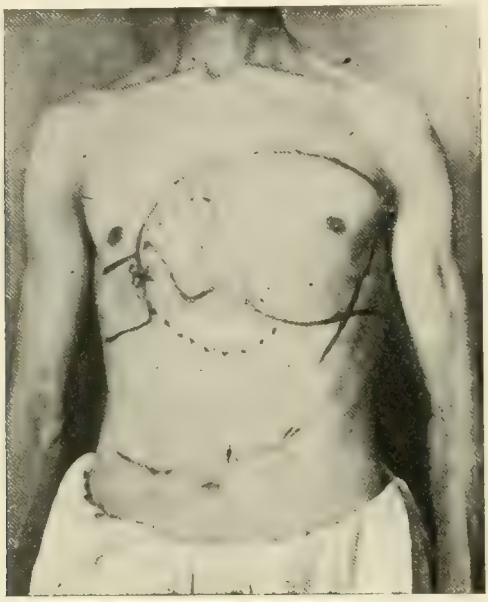
Heart.—There is no palpable impulse on the left side. There is an area of wavy pulsation on the right side in the 5th space. There is also a wavy pulsation in the epigastrium, especially between the ensiform and the left costal margin. The point of maximum impulse is in the 5th interspace 7 cm. from the mid-line on the right side. The sounds are best heard at this point. They are normal.

The abdomen is negative. The liver is palpable 4 cm. below the costal margin. The legs and the feet are not swollen. There is slight dulness of sensation below the knees, most marked in the recognition of heat and cold. There is a delay of about four seconds in the reflexes. There is no actual anæsthesia. There is little or no muscular incoordination of the upper extremities.

Eye examination.—Examinations of the fundus show no retinal changes. The left pupil reacts rather more sluggishly than the right.

June 23. The patient left the ward.

The urine is negative; no albumin is present. Temperature normal.



PICTURE REVERSED, RIGHT SIDE SHOWN TO LEFT.

The markings of the picture indicate the following:

The dotted line is the area of visible cardiac pulsation; the X the point of maximum impulse. The two parallel lines on the right side are the lines of relative and absolute hepatic flatness. On the abdomen is indicated the lower curvature of the stomach. On the left side is indicated the area giving the signs of pneumothorax.

This case was very interesting in more ways than one. He is a tabetic who evidently had pneumothorax fifteen years ago and remained in bed six months from the pain, weakness and shortness of breath which followed a foot race. During the past seven years he has heard a hollow ringing sound in his chest. On admission the signs of pneumothorax localized at the left base were definite. The great interest in the case lies in the association of the pneumothorax with tabes, and in the possible origin in over-exertion; but the duration is also of great interest. How long had the condition persisted; was it a recurrent case?

II. THORACENTESIS.

Cases of pneumothorax due to puncture of the chest one might think after a study of literature to be rare. Stokes,[79] however, admits by inference that it is not rare, for he says: "We know the entrance of air by the canula during paracentesis is a matter of trivial moment." Nevertheless Biach could find but three cases on record. The possibilities of such cases are several in number: (1) The tapping may wound the lung directly. (2) Air may enter through the canula if the technique be faulty. (3) The negative pressure exerted on the compressed and probably adherent lung may cause the rupture of the visceral pleura at the seat of the adhesions. (4) The negative pressure may be sufficient to cause the rupture of a superficial cavity. (5) The gas liberated by the reduced pressure from the blood in the pleural blood-vessels may enter the chest. This last case has received the name of "pneumothorax ex vacuo." (6) The local respiration of the tissues. (7) Rupture of an emphysematous bulla due to the negative pressure.

To discuss these cases briefly: Mayer[336] attributes the conception "pneumothorax ex vacuo" to West, but says Bard reported the first good case. He discusses all cases which could possibly be due to this cause, and finds but three and they not above criticism. Widal[216] attributes to See this same explanation for pneumothorax, viz.: the lungs being bound down are incapable of filling the pleural cavity and hence the liberation of gas from the blood. Senator[242] admitted the possibility, attributing the idea, however, to Bucquoy, but stated that the amount of gas in such cases must be small and hardly important. That gas can arise from the blood is only too evident from a study of the gas analyses which will be found under that heading, for if a pure gas be injected into the pleural cavity there will very soon be present a high percentage of CO_2 and of nitrogen, whose only origin could have been the blood. That the local respiration of the tissues can furnish gas is seen by the high percentage of carbon dioxide which gathers in the pleura after death. This, however, would not be any considerable element during life. The majority of the cases have been attributed either to the rupture of a subpleural cavity or to the tearing of the visceral pleura by an adhesion. In a few cases this has been demonstrated, nevertheless such are rare. The rupture of the subpleural cavity or the emphysematous bulla are both of them possibilities. That the needle itself may pierce the lung there

can be no doubt. Anders and McFarland [336] mention two cases of Herman Biggs caused by the puncture of the lung by a hypodermic needle, and Packard adds another, proven at autopsy, of fatal pneumothorax from the puncture of an emphysematous bleb. Our case No. 30 is a splendid illustration. Stengel added a case from paracentesis. Roy [337] reported the case of a girl 11 years old who was tapped for an old serous pleurisy. She coughed considerably. After the aspiration she expectorated a fluid very much like that aspirated and presented the signs of pneumothorax. They considered that this case was not due to trauma of the lungs, since no blood issued through the needle. Galliard considered that this case was not one of pleural effusion rupturing through the lung, but was a case of albuminous expectoration. Rendu asked if the girl might not be a case of healed pneumothorax in which the fistula was reopened by the aspiration, and mentions one case of his own in which this was the explanation. Phalip [322] reviews this subject at length. He considers that a weak place must always be assumed in the lung if the pleura is torn, since he says a simple prick is not enough. In the case of Herman Biggs and our own, however, this would seem to be false. He recommends that aspiration be done slowly and small amounts removed at a time.

We would also mention Villy's [321a] case, although we would not affirm that it belongs here.

CASE 30.—*Pyopneumothorax due to aspiration of a pyothorax; death; autopsy.*

Benjamin V., colored, age ? (is an old man). **Med. No. 2759.**

Admitted March 6, 1893.

Died March 10, 1893.

CLINICAL DIAGNOSIS.—*Pyopneumothorax. Pulmonary pleural and pericardial tuberculosis.*

COMPLAINED of "weakness and shortness of breath."

It was quite impossible to learn much of his FAMILY or PAST HISTORY. He is an oyster shucker by trade.

PRESENT ILLNESS.—This began about three months ago with cough and sharp pain in the right side. At first he expectorated freely, but now not at all. The pain is less; he coughs less but has grown weaker. He has chills at night.

PHYSICAL EXAMINATION.—The patient is lying on his left side; respiration 30 to the minute, pulse 96. Marked arterial sclerosis.

Thorax.—The right side bulges, movement is limited, the intercostal spaces are filled out. In the 1st and 2nd spaces on the right side the per-

cussion note is markedly Skodaic; below there is flatness without movable dulness. The breath sounds are noted as enfeebled above, absent below. The voice sounds are absent in the flat area. In the right back flatness begins at the spinal end of the 7th rib and extends upward in an ascending line nearly to the base of the spine of the scapula. Vocal fremitus is not entirely absent in this flat area in front or behind. The left lung is quite clear.

Heart.—Point of maximum impulse hard to determine, but apparently in the 5th i. s. just outside the nipple line. The sounds are clear.

Hepatic flatness extends to only about a finger's breadth below the costal margin in the mammary line. The abdomen is negative.

The patient was aspirated, 2000 cc. of a thin green pus obtained in which tubercle bacilli could not be determined. The aspirating was done at the 9th space and was followed by resonance to the 10th space in the angle of the scapula.

Respiration is feebly heard and the voice sounds are near the ear. In the erect posture there is resonance to the 6th rib in the mammary line in front. In the recumbent posture flatness begins at the 7th rib. The respiratory murmur is hardly heard below the second space in front. The pulse is rather feeble, 76 to the minute, with numerous intermissions. The heart sounds are now best heard in the fourth interspace an inch inside the nipple.

The urine contains a trace of albumin and hyaline and granular casts.

March 8. The right side is markedly distended; the intercostal spaces obliterated. The percussion note when the patient is recumbent is loud and tympanitic from the apex to the costal margin, but when erect begins at the 5th rib. In the back flatness nearly reaches to angle of the scapula. The respiratory murmur is heard only at the apex where it is very feeble, and the cough has a metallic ring. The coin sound and the Hippocratic succussion splash are well marked. The outermost point of cardiac impulse is in the mammary line.

March 10. "To-day there is no marked tympanitic quality to the percussion note over the right front nor is there any amphoric quality to the breath sounds even on coughing. The coin sound is of the most exquisite character. There is no metallic echo to the cough. Succussion is most marked."

The patient died to-day.

For the first four days the temperature was subnormal from 95 to 97° F. On March 8 it rose to 100° and was irregularly elevated till his death.

Autopsy No. 399.

Anatomical Diagnosis.—*Tuberculous pleurisy; pyopneumothorax; tuberculous pericarditis; old healed tuberculous lesions in apex of left lung, lower lobe. Bronchopneumonia. Chronic diffuse nephritis. Slight fatty degeneration of kidneys and liver. Brown atrophy of heart muscle. Pigmentation of liver and dilatation of central veins.*

Body 170 cm. long, old, emaciated man. Subcutaneous fat in small amount.

Diaphragm on right side at lower border of 6th rib, on left side at lower 5th.

Right pleural cavity contains 2200 cc. of thick turbid pus. The lung is compressed and occupies the upper, back and internal parts of the pleural cavity. It is densely adherent to the diaphragm below, to the superior part of pleural cavity above, and behind to the posterior wall of thorax. On opening the pleural cavity there was no evidence of escaping air, but on introducing a canula into bronchi and filling chest with water, air escaped on blowing through the canula at about the middle of the lower lobe. A small probe could be passed into the lung at this point, but it does not enter a cavity, and on section no cavity is found in the lung. The pluera at this point is thin and apparently deficient. The pleura covering the lung is much thickened, opaque, greyish-yellow color and not uniform. It is smooth along the posterior border, but at the base over anterior half is rough, irregular and worm-eaten in appearance. The lung on section is dense and deeply pigmented, the bronchi dilated, thin and contain sticky mucus.

Left lung is bound down by adhesions to the diaphragm and chest wall, those to the diaphragm are very firm and inseparable, the others are broken down with difficulty. The lung is voluminous, pleura pigmented, the apex is retracted and dense caseated tissue is present in this part, a single calcareous nodule the size of a bean occurs in the lower lobe about its middle. On section the surface is dark, injected, much blood-stained, and serum can be squeezed from it. In apex, adjacent to retracted area is an emphysematous condition. Bronchi contains mucus and frothy serum. The posterior aspect of the upper lobe near base contains a consolidated nodule the size of a pigeon's egg, in the center of which is a dilated bronchi. It is smooth, slightly granular and greyish-red in appearance. "This is probably a case of primary pleural tuberculosis with secondary involvement of the lung and pericardium."

In this case the patient had had signs of pleurisy with effusion for about three months. Following the aspiration at which time 2000 cc. of pus were removed, the right side was at once found to present signs of pneumothorax which the next day were marked. It became distended, hyperresonant on percussion, with extreme movable dulness. The breath sounds were not heard. The coin sound was obtained and the succussion splash was well marked. The patient died four days later.

At the autopsy an interesting feature was found. The right pleural cavity contained 2200 cc. of pus. No air could be heard to escape when the cavity was opened, but on putting a canula into the bronchus the air was easily blown through a perforation at the middle of the lower lobe. This perforation was in a part of the

pleura elsewhere much thickened but here worm-eaten and deficient. It did not communicate with the cavity of the lung.

The interesting point of this case is concerning the cause of the pneumothorax. It was doubtless caused by the aspiration. Nevertheless at autopsy a perforation of the lung which was previous to air blown from the bronchus was demonstrated. The condition of the pleura would indicate that the empyema was about to perforate through the lung. Hence this cause of pneumothorax was also present. The aspiration may have hastened the process, by rupturing in the opposite direction. Taking the position of the lung into account it is quite impossible that the aspirating needle should have wounded the lung; this the negative tension after aspiration of 2000 cc. could do; or we may assume that the pulmonary fistula developed later and the air entered from externally.

At any rate the fistula described would have been a valvular one and it is interesting that the breath sounds were not audible.

CASE 31.—*Pneumothorax due to aspiration of a pyothorax; death.*
Harvey, C. L., aged 24. **Med. No. 5217.**
Admitted August 8, 1895.
Discharged August 24, 1895.
DIAGNOSIS.—*Pleurisy with hæmorrhagic effusion.*
The patient COMPLAINS of pain in the left side and dyspnœa.
The FAMILY HISTORY is good; no tuberculosis.
PAST HISTORY is good. Six years ago he had "malaria" but without distinct chills. He had a left-sided pleurisy twelve years ago. Was in bed for four weeks. He has never had pneumonia or rheumatism; has not been subject to cough since the pleurisy. His habits are good.
PRESENT ILLNESS began Christmas, 1894, with quite marked shortness of breath, increasing much on exertion. He had noticed slight dyspnœa for the four months previous to this. He did not have any pain in the chest at this time nor was he confined to bed. During the past eight months the dyspnœa has increased to the present time. He has a dry, hacking cough whenever he exerts himself; there is no expectoration. During the past three weeks the patient has complained of a dragging pain in the lower left axilla and left front of the chest. He has had no fever. He has had a great deal of palpitation of the heart on exertion. The appetite is poor. Has lost ten pounds since Christmas. He has been working until the present date.
PHYSICAL EXAMINATION.—The patient shows a slight dyspnœa; pulse 96. The thorax is somewhat barrel-shaped. Expansion decidedly limited on the left side.
The lungs.—Percussion note on the right side is clear to the third

space. Below this is deficient resonance. In the right axilla the note again is clear. Over the left front is absolute flatness from the clavicle, extending out into the axilla and lost in the area of cardiac flatness. Vocal fremitus over the left side is practically gone.

On auscultation over the right front above the 3rd space and in the right axilla the breath sounds are normal. Below this they are somewhat enfeebled and distant. Over the left side and throughout the left axilla the breath sounds are scarcely to be heard; they are very feeble and distant. The voice sounds everywhere are distant and nasal.

Backs.—The percussion note on the right side is clear throughout. On the left side there is complete flatness. The vocal fremitus, though not entirely absent on the left side, is very slight. On auscultation of the right back the sounds are normal, although harsh and exaggerated. On the left they are almost absent which is also true throughout the left axilla. The breath sounds are nowhere tubular.

Heart.—The point of maximum impulse is in the 4th space just in the nipple line of the right side 11 cm. from the mid-line. The relative dulness extends from the juncture of the first space with the sternum outwards to the point of maximum impulse. Below it is lost in the hepatic flatness. There is quite plain visible pulsation over the right chest, overlying the relative dulness to the right of the sternum. Hepatic flatness does not extend quite to the costal margin. The spleen is easily palpated.

August 9. The left pleural cavity was aspirated; 2660 cc. of very bloody fluid obtained. The needle was introduced in the 9th space in a line with the angle of the scapula. The patient coughed much during the aspiration and complained of severe dragging pain over the præcordium.

Microscopical examination shows many red blood corpuscles.

August 10. The patient is lying on his back; pulse 96. The thorax is symmetrical as the patient lies still. The left side, however, moves but little. The cardiac impulse is not visible. Hyperresonance is present throughout the right side. Flatness in the left front at the 4th rib in the mammillary line though not until the 7th rib in the mid-axilla. The cardiac dulness does not pass the left sternal margin. On auscultation the respiratory sounds are clear in the left upper front, becoming distant and almost flat in the dull area. Fremitus almost absent. The heart sounds are best heard in the 4th interspace 2 to 3 cm. inside the nipple line. When the patient lies on his left side there is flatness to a point a little above the angle of the scapula. Vocal fremitus is not entirely absent though it is diminished. Respiration is distant and of a tubular quality. Vocal fremitus is diminished but not absent.

August 12. There is flatness at the 3rd rib in the erect posture; at the 4th in the recumbent. The heart sounds are well heard to the left of the sternal margin.

August 14. Flatness begins at the upper border of the 5th rib; changes hardly any on movement.

August 24. Discharged.

Urine negative. Temperature irregularly elevated from normal to 101. It remained normal after August 13.

Second admission, on February 27, 1896. Med. No. 5849.

Discharged March 14, 1896.

DIAGNOSIS.—*Pleurisy with effusion; pneumothorax.*

The patient returns to-day complaining of shortness of breath. Looks well. He has lost, however, seven pounds since last in the hospital. The thorax as the patient lies quietly looks a little fuller on the right side than on the left, and expands more on the right. The point of maximum impulse is seen inside the left nipple line. On percussion the right side is clear from the apex to the base. On the left side the note is high pitched and tympanitic at the apex, becoming exceedingly dull at the 3rd rib and nearly dull in the axilla. Vocal fremitus is absent.

Backs.—Percussion is clear over the right side. On the left side the note is very dull, almost flat throughout. Vocal fremitus, though diminished, felt everywhere except below the angle of the scapula. There is no difference in the line of dulness in the recumbent and erect posture. The point of maximum impulse is 11¼ cm. from the median line; the sounds are clear. Hepatic flatness extends 3 cm. below the costal margin on deep inspiration.

February 28. Dr. Osler: Palpable difference of expansion is very marked; on percussion below the 2nd rib in front there is flatness. Behind flatness from the spine of the scapula to the base. Mobility is impaired. Tactile fremitus is not obliterated. There is no enlargement of the glands; no thickening of any of the bones; no clubbing of the fingers. On admission a hypodermic needle was inserted in the left side at the angle of the scapula and a syringe full of hæmorrhagic fluid was obtained.

March 2. Dr. Osler: The left shoulder is lower than the right. Cardiac impulse is visible outside the nipple line on the left side. On percussion over the left back there is flatness. Tactile fremitus is nowhere obliterated, but is diminished. The breath sounds are feeble over the left side and heard only on deep inspiration. The left pleural cavity was aspirated to-day. 1180 cc. of a very hæmorrhagic fluid were obtained. The patient did not begin coughing until 900 cc. were removed. The patient complained little.

March 3. This morning there is dull tympany in the front and to the 6th rib in the mid-axilla. Cardiac impulse, which before the tapping was 11¼ cm. from the mid-line, is now with difficulty made out. Tympany reaches in the 4th and 5th spaces to the parasternal line, while the heart sounds are best heard in the 4th space 4 cm. inside of the mammillary line. The heart has therefore moved considerably to the right. Vocal fremitus is well felt at the apex; respiration is feeble at the apex, becoming practically absent as one reaches the axilla. The resonance and the vocal fremitus extend to the angle of the scapula. The respiration is feeble. Below the angle of the scapula there is flatness and distant respiration.

March 4. At seven o'clock to-day the patient had a severe shaking chill. The temperature rose to 105.9. Since last night has had pain in the left front of the chest which this morning has grown more severe. It prevents his taking a deep breath. This morning there is slight retraction of the left side which moves little with inspiration. Tympanitic resonance throughout the left front and axilla. In the erect posture there is flatness. Over the back there is resonance to the 8th space. Vocal fremitus is increased in the upper left front and felt distinctly in the left side and back.

Heart.—Point of maximum impulse is not seen. The flatness occupies a small area between the 4th and 5th rib to the left of the sternum. Respiration and resonance on the right side are clear. On the upper left front the respiration is enfeebled. The voice sounds are increased in intensity. As one passes downward into the axilla and lower chest both respiration and voice sounds become diminished in intensity. Throughout the back the respiration is enfeebled. The voice sounds are increased in intensity until one passes below the 8th rib at which point both are enfeebled. There is no characteristic coin sound although the sound is intensely heard.

March 5. The patient is more comfortable. The left side to-day looks a little fuller than it did yesterday. The apex beat is not visible or palpable. There is high-pitched tympany over the whole left front reaching in the recumbent posture to the 5th cartilage in the parasternal line, to the 6th rib in the mammillary line, to the 7th rib in the mid-axillary line. Cardiac dulness reaches to the right of the sternum. There is no movable dulness. Over the upper front respiration is rather enfeebled; expiration is prolonged. Below it becomes very feeble and is scarcely heard in the axilla. The voice sounds are quite loud above and feeble below. Vocal fremitus is increased in the upper left front. The back is dull throughout. Below the angle of the scapula it is almost flat. Vocal fremitus is increased above but is felt throughout. The coin sound is suggestive. There is no amphoric breathing; no metallic tinkling.

March 7. The area of flatness is practically the same in the recumbent posture. There is nowhere amphoric respiration. There is resonance behind to the angle of the scapula. Vocal fremitus faint at the base. There is no succussion splash.

March 12. The sounds are heard much better on the right side of the sternum. Flatness in the recumbent posture is just outside the nipple at the 5th rib. There is no movable dulness; no characteristic coin sound.

March 14. The patient is better but still not well enough to leave the hospital; he insists, however, upon going.

The urine is negative. The temperature was normal until March 3, when, with a chill, it arose to 105.9°, and then gradually fell reaching 100° on March 14.

Third admission, September 15, 1896. **Med. No. 6631.** He was at once transferred to the surgical side. The patient returned with pleural

effusion. Since leaving the hospital he has improved in general health but has noticed a reaccumulation of fluid on the left side. When lying down he has noticed a splashing sound for the past six weeks. Since then he has been worse, with shortness of breath, and steady cough.

Physical Examination.—The patient looks somewhat emaciated. There is no dyspnœa. The left side does not move as much as the right. The intercostal spaces are equal. The clavicles stand out prominently. The left shoulder is a little lower than the right. The left side is a little flatter. As yet there is no curvature of the spine. The intercostal spaces on palpation are very close. Tactile fremitus over the left upper front is distinctly less; it is lost below the 5th rib. It is felt over both bases but lost in the left side. There is distinct pulsation just below the right nipple in the 4th interspace. It is also seen in the 3rd and 5th spaces. A pulsation is seen in the left infra-mammillary region, entirely below the 5th rib. It extends into the 6th space and a finger on the 7th space is distinctly lifted by it. It extends below the posterior axillary line. The limits behind and below are as follows: The mid-axillary; the upper border of the 6th rib behind to the posterior axillary fold, the impulse gives the impression of a good deal of vigor. Percussion over the right side is normal. There is defective resonance at the left apex, anteriorly shading at the lower margin of the 3rd rib into absolute dulness which extends into the axillary region when the patient lies down. In a sitting posture the dulness reaches as high as the 2nd rib. There is flatness from the base to the apex posteriorly on the left side except for some slight resonance in the upper interscapular region. There is pneumothorax in the upper and mid-axilla on the left front. Auscultation over the left side behind:—the breath sounds are everywhere feeble; at the extreme base posteriorly they are audible; at the apex small crackling râles; in the upper interscapular region the breath sounds are louder, piping and medium sized mucous râles are heard. On deep inspiration in the infrascapular region there is distant tubular breathing. On coughing no splash is heard. There is succussion when the patient lies on the right side, yields a loud splash, very superficial. The coin sound is very distinctly transmitted on the left side. The sputum is mucopurulent, contains no elastic tissue and no tubercle bacilli.

The urine contains a trace of albumin.

Surg. No. 5840.

Transferred to Surgical Side Sept. 17, 1896.

Discharged Oct. 6, 1896.

On Sept. 17 Dr. Bloodgood excised portions of two ribs and evacuated the pus present. There is no note of the condition found in the pleural cavity. He left the hospital much improved but was reported to have died Jan. 1, 1898.

The patient was in the hospital three times. The first time with left-sided pleurisy with effusion. It is an interesting question whether

the aspiration of 2660 cc. from the left side August 6, caused pneumothorax, for some of the most important signs are present; the maximum impulse of the heart before aspiration was under the right nipple; after the removal of so large an amount instead of moving far to the left, the heart remains entirely to the right of the median line. The side is hyperresonant yet the breath sounds are distant and of a tubular quality and the vocal fremitus was diminished but not absent. Such a remarkable picture can best be explained as pneumothorax. He left to return six months later with the effusion reaching the 2nd rib, and was tapped. Following the aspiration of 1180 cc. the signs of the left side changed much. Instead of flatness was dull tympany with extreme movable dulness at first but noted later as absent. The heart moved considerably to the right and its area of dulness diminished. The breath sounds were quite absent below the apex, and voice sounds increased. The coin sound was only suggestive. There was no metallic tinkle, no succussion splash. The fever rose with a chill and remained elevated. We suspect these signs are best explained by air in the pleura the result of the aspiration and subsequent consolidation of the lung. Six months later however, he returned with pneumothorax, for even he could hear the splashing. The coin sound was distinct, the dulness movable, yet the breath sounds distant.

Should this diagnosis be correct, pneumothorax and consolidation the result of injury of the lung at aspiration, the signs would be well explained for the succussion splash and the coin sound would not appear until the lung had resolved, allowing more room in the chest The distant breath sounds are quite in accord with the other cases of pneumothorax from aspiration.

CASE 32.—*Hydropneumothorax of the right side due to aspiration of a hydrothorax; recovery.*

George L. M., aged 50, colored. **Med. No. 6723.**

Admitted October 7, 1896.

Discharged December 4, 1896.

Clinical Diagnosis.—*Pleurisy. Pneumothorax.*

Complains of "asthma" and disordered stomach.

Family History.—Negative. No tuberculosis.

Past History.—"Asthma" one year ago; otherwise negative.

Present Illness.—For one year the patient has suffered from irregular attacks of dyspnœa, which in July, 1896, became more frequent and severe. He has had to remain at home. He complained of a soreness

over stomach when he walked as if his heart was going to burst. His dyspnœa is intense. There is no pain in the chest, no cough. He expectorates some mucus. He occasionally expectorates blood when he has a cold but never vomits blood. No night sweats; no headache; has had swelling of the feet twice during this attack and has lost 28 pounds of flesh.

Physical Examination.—The patient lies on the left side. Respiration has something of the Cheyne-Stokes character, 40 to the minute. Pulse, 92; tension not increased; regular in force and rhythm; considerable arterio-sclerosis. Thorax rather barrel-shaped. The left side moves more on respiration than the right. Apex beat is visible in the 6th space 12¾ cm. from the midsternal line.

Percussion.—The left side is clear throughout. On the right a distinct tympany is obtained at the apex becoming very marked in the third space; flat at the 5th rib. Movable dulness obtained but the vocal fremitus is absent in that area. In the back there is flatness at the right base to a point 6 cm. above the angle of the scapula. Respirations are harsh and puerile throughout the left lung accompanied by occasional sibilant râles. Above at the extreme right apex it becomes enfeebled; as one passes down and in the 4th interspace a soft friction is heard; also fine moist râles are heard in the 4th and 5th space up to the axilla. Below respiration is enfeebled and of somewhat tubular modification with fine crackling râles on inspiration. In the back numerous fine moist râles are heard throughout the left side. On the right side voice sounds are muffled but do not sound as far away as is usual in a pleurisy.

October 9. Dr. Osler: There is flatness on the right side. The interspaces are here less distant. Tactile fremitus diminished; distinct movable dulness. The percussion note is somewhat Skodaic and the movement is less marked.

October 20. Patient is aspirated this morning, 800 cc. of a cloudy, bloody fluid. On the 22d patient feels better. Left side of chest still moves more than the right. The maximum cardiac impulse is 11 cm. from the midsternal line. In the semi-erect posture flatness begins at the 5th rib. The dulness is movable.

November 1. An unsuccessful attempt was made to aspirate the patient. On the following day the left side of the chest moves much more than the right, which is almost motionless. The left side of the chest looks fuller and moves more than the right. Respiration during the night has been rapid, 40 to the minute. Vocal fremitus is diminished everywhere on the right side in the right axilla. In the right axilla there is subcutaneous emphysema extending to the apex of the axilla and to a certain distance over the back. Point of maximum impulse 12½ cm. from the midsternal line. On percussion the note is clear throughout the left front and axilla. The right front is a little tympanitic at the apex. Passing downwards resonance becomes loudly tympanitic, becoming flat in the recumbent posture at the 8th cartilage in

the mammillary line; practically no movable dulness. In the back on the left there is normal resonance and on the right side relative dulness.

Auscultation on the left side.—The breath sounds are rather exaggerated and harsh. In the lower part of the axilla a few crackling râles are heard.

Right side.—At the extreme apex respiration is enfeebled; slight tubular modification; occasional crackles on inspiration. Below the second rib respiration is distant amphoric with an occasional metallic tinkle. the coin sound, though not loud or clear, has a metallic resonance. At the 3rd interspace in front exquisite succussion is heard when the patient shakes himself.

November 13. The succussion not obtained.

On the 4th of December the patient leaves at his own request.

While in the hospital his temperature was subnormal for the most of the time, though irregular and sometimes reaching 100°.

Bacteriological examination of the aspirated fluid was negative. No tubercle bacilli found in the sputum.

The sequence here is only too evident. A patient with right-sided hydrothorax was aspirated unsuccessfully and on the next physical examination the signs of pneumothorax were present; the subcutaneous emphysema, the clear percussion note where had been flatness, the distant amphoric breath sounds, the metallic tinkling, the coin sound, and the succussion splash.

The fluid now could seek a deeper level since the lung collapsed and the diaphragm sank.

Thirteen days later the succussion splash could not be heard.

CASE 33.—*Hydropneumothorax resulting from aspiration of a hydrothorax; death; autopsy.*

John V., aged 55, white. **Med. No. 8502.**

Admitted April 15, 1898.

Died May 27, 1898.

CLINICAL DIAGNOSIS.—*Nephritis, chronic arterio-sclerosis, mitral stenosis and insufficiency.*

He came in COMPLAINING of "swelling up all over and inability to get his breath."

FAMILY HISTORY is negative.

PAST HISTORY.—Is ignorant of any diseases of childhood. He had yellow fever at thirty years of age while in the West Indies and rheumatism twenty years ago. He has been a very heavy drinker, smoker, and habits in general have been bad.

PRESENT ILLNESS began four weeks ago. He noticed swelling of the feet and legs which increased on walking, less at night, not painful. Two months ago began to get short of breath. This came on very sud-

denly and increased to orthopnœa. The orthopnœa disappeared in two weeks but has recurred at intervals since. Eight weeks ago had swelling in the arms and abdomen. During the past two years he has noticed that the urine passed has been diminished in amount but the frequency increased. He has been treated at the dispensary for four months. His present admission follows a debauch of about a month's duration, during which his condition grew worse.

Physical Examination.—Temperature, 98.4; pulse, 112; respirations, 26. Patient is cyanotic, but not anæmic. Both arms are reddened and œdematous. The pulse is small, rapid, not easily compressible; the artery is not palpable. The respiratory movements are equal on both sides. Respiration is of the Cheyne-Stokes type. Vocal fremitus is marked over the left front, axilla and back. Over the right side it is indistinctly felt; over the front as far as the 4th interspace. Below this it is not felt.

Percussion.—Over the entire left side the note is hyperresonant. Over the right front above the fourth space the note is dull; below flat over an area corresponding to the loss of vocal fremitus. Over the right back there is dulness. Here the dulness on the right side is movable one interspace.

Auscultation.—Over the left front, crackling râles are heard to the axillary line. The breath sounds are harsh, prolonged on expiration. In the axilla they are the same. Over the left back above the level of the spine of the scapula the sounds are not well heard. Below they are harsh with numerous moist dry râles. Right side.—Sounds above the flat area described are poorly heard; below they are heard but very faintly. There are no adventitious sounds.

Heart.—Point of maximum impulse is palpable in the 7th interspace 12 cm. from the midsternal line. The dulness begins at the 3rd rib. The sounds at the apex are clear. The pulmonic second is accentuated. At the apex a faint systolic murmur is heard.

Abdomen is much distended; movable dulness. The liver dulness extends to 1 cm. above the costal margin. The spleen is not palpable. The legs are very œdematous.

April 17. The œdema was markedly reduced under strong purges. Small spots of hæmorrhage are now seen over the flexor surfaces of both forearms and elbows. Leucocytes 12,000.

April 18. The right chest was aspirated and 1700 cc. of fluid removed, of a clear yellow color. Cultures were negative.

April 19. The patient is cyanotic. Respiratory movements are shallow; intercostal spaces are obliterated.

Percussion.—The chest is resonant and equal over both upper fronts. Below the 4th line on the right side one gets absolute flatness. On the left side the note gets slightly dull in the left axilla. The dulness is movable one space on the right side. Posteriorly the right lung is dull to the angle of the scapula and on the left side below the level of the 8th dorsal spine.

Auscultation.—Over the areas of dulness the breath sounds are almost entirely absent. An occasional râle is heard. Over both upper fronts the sounds are those of an emphysematous lung; expiration is prolonged; an occasional piping râle is heard in the axillary region and trickling of water on deep inspiration with metallic tinkling. Respiration is somewhat labored.

Heart.—Cardiac dulness is obliterated. The point of maximum impulse is neither visible nor palpable. The sounds are best heard in the 5th space 12 cm. from the midline. A soft diastolic murmur is heard along the left sternal border. The liver dulness extends four fingers' breadths below the costal margin. The edge is well felt. The spleen is palpable on deep palpation. The abdomen is full but undoubtedly contains fluid; is dull in both flanks. There is some œdema of the legs. The skin is very tense. The patient is noisy, restless and excited when spoken to, but otherwise seems to be of a clear mind.

April 19. The patient is better this morning. He takes his sweat baths quite well. One hour after the sweat bath of yesterday he became quite cyanotic, pulse becoming a trifle weak and the mental condition worse.

April 23. The patient's right chest was again aspirated and 1500 cc. of a clear straw-colored fluid with a specific gravity of 1006 were obtained. The fluid contained only a trace of urea. Before aspiration the dulness extended up in front as high as the 3rd space and behind to the lower angle of the scapula. The breath sounds are not well heard in the front or in the back. After aspiration, however, no resonance was obtained down to the 6th rib and behind only relative dulness could be obtained throughout. The patient's heart is slightly accelerated. He had one or two coughing paroxysms during the aspiration, but otherwise seemed much relieved.

April 25. The patient is rather short of breath this morning, but is otherwise doing well.

May 2. The patient is almost entirely irrational. The breathing is labored. Flatness begins at the 3rd intercostal space on the right side in front. There is dulness over the back up to the angle of the scapula. The breath sounds are not heard behind. The examination is difficult on account of the great œdema and restlessness of the patient. There is extreme cyanosis of the feet and hands. There are cyanotic patches over the legs, arms and trunk. A systolic murmur is well heard at the apex. This morning the patient was aspirated and 1860 cc. of clear, slightly turbid yellow fluid was obtained. There was some coughing during aspiration and the most extreme Cheyne-Stokes respiration. Following aspiration the cyanosis becomes much less, and breathing is less labored. He answers questions rationally. The feet are cyanotic but not so blue. The pulse is slower but more regular. The aspirated fluid had a specific gravity of 1006 with a very small amount of urea.

May 6. Aspiration was performed and 2000 cc. of a blood-stained fluid were removed. The patient was immediately relieved, becoming almost rational. The dulness after aspiration extends nearly to the 4th interspace.

May 14. Aspirated again to-day. 1850 cc. of clear fluid were removed. Some air sucked into the pleura through the aspirating needle at first, and although much was aspirated out afterwards the resonance after aspiration extended to the 7th rib on the right side. The patient is feeling more comfortable. Cyanosis decreased; respiration is less labored. The pulse is rather small and thready.

May 18. The patient is very much cyanosed and weaker. The note over the right side is still resonant. There is well-marked movable dulness extending up to the posterior axillary line. The breath sounds are distant and obscure.

May 21. Aspirated at the right back. 580 cc. of blood-stained fluid removed.

May 27. The patient has been much weaker. He does not talk on account of the dyspnœa. He is irrational. The fluid is again increased on the right side. Dulness is evident on the left side to the mid-axillary line suggesting the presence of fluid there.

May 28. The patient has been very cyanotic lately. Slight scratches develop into open sores so that the hips and flanks are covered with excoriations. There is an ulceration on the lower lip and the tongue is the seat of three deep ulcers. The hands, feet and face are quite blue. Respirations are very enfeebled and distressed. Death occurred quietly without convulsive movements.

The urine while the patient was in the hospital was always scanty in amount, seldom as much as one liter being recorded. Specific gravity for the most part was from 1011 to 1015. A great deal of albumin was present with granular, epithelial, hyaline, blood and pus casts. The urea was diminished varying from 2 to 13 grammes a day, although the total amount is hard to reckon, since considerable urine was lost. The Diazo test was not positive. The temperature was practically normal until the aspiration of April 23, after which it rose to 100.4° and remained slightly elevated for four days. After the aspiration of May 2 it rose from normal to 101.6°, but fell at once to normal, where it remained with few rises to 99° to 100° until May 26, by which time it became subnormal, not rising above 97° on the day of death.

Autopsy No. 1099.

Anatomical Diagnosis.—*Chronic diffuse nephritis; red granular kidneys. Arterio-sclerosis. Chronic proliferative endocarditis (mitral stenosis). Hypertrophy and dilatation of heart. General chronic passive congestion. Atheroma of aorta, coronary arteries, basilar and other peripheral arteries. Abnormal position of large intestine. Hydrothorax. Ascites. Chronic adhesive pleurisy. Pneumothorax, right side.*

Body of a medium-sized white man showing general anasarca. Skin sallow. Abdomen distended. Rigor-mortis well marked. All the tissues are œdematous.

Right pleural cavity shows presence of small amount of air (water test). It contains besides a large amount, 1000 cc. of almost clear serous fluid.

There are firm, thick bridges of connective tissue binding upper, middle, and upper part of lower lobes to the thorax. Left pleural cavity contains 1000 cc. of clear straw-colored fluid. No marked adhesions between its layers.

Lungs are contracted. Right adherent as described. Both are uniformly congested, of increased consistence containing air throughout. On section they present the typical picture of deep brick-red color. Surfaces are smooth and dry. At bases behind, tissue is deeper in color, beginning hypostatic congestion—chronic passive congestion. There is considerable coal pigment diffusely scattered through parenchyma. The bronchial mucous membrane is congested.

Præcordial space is extensive owing to increase in size of heart and retraction of lungs. Heart weighs 600 grammes. Right ventricle is somewhat dilated. Tip of left auricular appendage just visible on left side. Apex formed entirely by left ventricle.

This case of nephritis and cardiac vascular disease with hydrothorax was repeatedly aspirated, large amounts—1800 to 2000 cc.—being removed each time. On one occasion the air was allowed to enter through the needle, probably not much, for the signs did not much change. It is of interest that the breath sounds remained distant and obscure. It would be hard to say that the air made the prognosis more unfavorable.

CASE 34.—*Pneumothorax of left side, pure, due to puncture of the spleen; death from appendicitis; autopsy.*

Mrs. M. J., aged 35, white. **Med. No. 8512.**

Admitted April 18, 1898.

Died May 17, 1898.

DIAGNOSIS.—*Meningitis serosa; acute purulent appendicitis; pneumothorax from puncture.*

Patient COMPLAINED of weakness and headache.

FAMILY HISTORY is negative.

PAST HISTORY is negative.

PRESENT ILLNESS began two weeks ago with a cold chill, not shaking. The chill lasted a day and a half, followed by sweating. She was then out of her mind for nine days. Since then she has been as at present. She has general soreness, headache on the top of her head, sometimes very acute; at other times only a dull pain; has no appetite; there is no swelling of the feet or legs; there is general weakness.

PHYSICAL EXAMINATION.—Patient is lying flat on her back with a dull expression. Respiration is slightly noisy and labored.

Thorax.—The expansion is good and equal. The percussion note is resonant everywhere. The respiratory sounds are normal. There is no adventitious sound.

Abdomen.—The walls are lax. Liver dulness begins in the 6th rib in

the mammillary line and extends 3½ cm. below the costal margin. It is soft everywhere on palpation.

April 21. On April 19 the patient had a shaking chill; another one on April 20. The temperature rose to 105.5° and 104.5° during the respective paroxysms. During the height of the fever the pulse was dicrotic but only 100 to 108 per minute. The patient was in a state of partial delirium. On the morning of April 20 the spleen was readily palpable. This morning it is not so well felt. The patient resists examination. The blood is negative for malarial organisms. Vaginal examination is negative.

April 23. At 4.30 p. m. the spleen was aspirated. The blood obtained was aspirated for malarial organisms but was found negative. The patient complained considerably during the remainder of the afternoon of pain in the left side. At 7.30 p. m. the whole left chest was hyper-resonant. The breath sounds are very feebly heard. Over the middle of the left back they were not heard at all. The breath sounds in the right chest are harsh and abnormally loud. At 10.30 p. m. vocal fremitus is absent on the left side; not very distinct on the right. Heart impulse is felt only over the xiphoid; not at all in the usual cardiac area. There is no emphysema in the tissues about the point of puncture.

April 24. Dr. McCrae: Temperature at 8 a. m., 100 degrees. The patient is lying quietly with the same stolid expression. Respirations 24, not labored. There is not the slightest dyspnœa. Pulse is 80, good in force and rhythm. The note over the fronts is resonant; that over the left front is hyperresonant. Over the left front the breath sounds are heard very distantly and faintly. The note in the upper left axilla is resonant and of much the same pitch as in the front. At about the 7th interspace in the mid-axillary line it becomes tympanitic (probably stomach tympany).

Backs.—Over the left back the note is more resonant than on the right back. The breath sounds are heard over the right back; they are clear. They are distant and feeble over the upper left back and not to be made out below. There is no visible heart impulse. The sounds are equally well heard over so wide an area that the point of maximum impulse is not determined. The area of cardiac dulness is gone. Liver dulness begins at the 7th rib. There is no abdominal distension, no tenderness. The patient holds herself stiffly while being examined.

April 24. Dr. Osler: General condition is good; pulse quiet. There is a drop in the temperature from 102° at 6 a. m. to 97° at noon. This afternoon it is 97. The patient is reclining on the right side. The apex beat is neither visible nor palpable. Cardiac impulse lifts the lower sternum. There is rather a hyperresonant note over the left apex and upper axilla as low as the 5th rib with a flat, slightly tympanitic quality below this and a markedly tympanitic note to the 10th rib. The note is perhaps better termed "remarkably full instead of tympanitic." Anteriorly this extends nearly to the costal margin in the 6th and 7th spaces and to the 10th rib in the mid-axillary line. On auscultation with quiet breathing there is nothing heard in the axilla excepting a little rustle in expiration.

There is nothing audible in the infraclavicular region on quiet respiration. Along the left sternal margin is well marked respiratory murmur. In the mammillary region there is the same silence. "The silence on quiet breathing over the front and side of the chest is remarkable, especially when contrasted with the opposite side." On deep inspiration there is feeble distant amphoric breathing in the infraclavicular and lower axillary region. The note has a flat tympanitic quality behind and the breath sounds are indistinctly amphoric.

April 26. The patient was delirious during the past night. This morning the respirations are slow, somewhat noisy. Otherwise they are much the same as before.

April 27. On vaginal examination nothing could be found to account for the temperature or the symptoms. The patient is lying on the right side, dull, with the eyes closed, and pays no attention to questions. Pulse is 104. There is a somewhat dusky cyanotic hue to the skin. On the left side the chest looks rather full, distinctly tympanitic on percussion. The cardiac dulness reaches 6½ cm. to the right of the mid-line at the 5th rib. The patient is breathing quietly. A feeble distant respiration is heard in the front and axilla on the left side.

April 29. The patient looks bright and rational this morning for the first time for three days. Breath sounds are heard over the left front and distinctly on the left back. A slight frictional rub is heard as yesterday just in front of the point of puncture after aspiration of the spleen.

April 30. The patient was found lying with the knees drawn up. The abdomen is everywhere soft; there is no tenderness.

May 1. The patient has been semi-conscious all day.

May 2. The patient is dull and cannot be roused. The pupils are negative. The note in the right axilla has a flat tympanitic quality and the breath sounds are hardly to be heard. The breath sounds are feeble over the left front. The note is almost tympanitic over the lower back, at which point the breath sounds are feeble. There is no abdominal distension.

May 4. This morning the patient is lying in a dull condition. The head is held rigidly so that any attempt to raise it raises the shoulders. The left side of the thorax does not look as full as before. The extreme tympany is not as marked in the upper part of the chest, although it is still tympanitic in the lower part. Respiratory murmur is feeble over the left side, but clear on the right.

Heart.—Relative cardiac dulness begins 6¼ cm. to the right of the sternum. The abdomen is negative. Lumbar puncture was done, but cultures from the fluid were negative.

May 7. Dr. Osler: The breath sounds are now heard on the left side. Respirations are slow, 9 to the minute. Pulse, 120, well-marked feeble rhythm. Pupils are negative. The head is thrown back a little. The patient winces a little when it is moved.

May 9. Patient had a profuse sweat last night. Lumbar puncture was negative.

May 11. The temperature has been normal for two days. The neck is still rather stiff, but less so than formerly. The left side of the chest moves considerably with respiration.

May 12. The patient shows symptoms of active delirium and difficulty in swallowing.

May 16. During the past few days much the same condition has obtained. The abdomen is still negative. To-day the patient has grown much worse and is failing rapidly. The pulse is about 160 to the minute and irregular. The rhythm is fœtal. There is marked tremor, especially of the arms. Respirations 68; very shallow. The patient is perspiring profusely. The eyes are negative. She died this night.

The leucocytes since admission have ranged from 10,000 to 16,000 per cm.

The temperature during the first seven days was very irregular, from normal to 105.5°. From April 26 to May 2 it was constantly elevated, ranging from 100° to 102°. It then fell to normal. There were slight rises until May 12, when it gradually rose to 105.8° on the day of death.

Autopsy No. 1097.

Anatomical Diagnosis.—*Acute purulent appendicitis. Pneumothorax. Chronic adhesive pleuritis. Cyst of parovarium.*

Body of woman, short stature, somewhat emaciated, sallow skin. Pupils equal, middle dilated, no arcus. Thyroid shows enlargement (bilateral). Small cystic nodule over the right side of lower jaw. Breasts normal, fully developed. Linea albicantes over lower abdomen. No general gland enlargement. No œdema of ankles. Slight rigor-mortis. Beginning bed sores over sacrum. Abdomen not distended.

Diaphragm on left side is very low. Before opening thorax its highest point on left side was 6th rib, on right side 5th space. On further examination the left attachment of diaphragm is almost at costal margin. On the right side the attachment is highest. The transverse oblique position of diaphragm is striking.

Left pleural cavity contains air (water test). After removal of sternum the left lung is found retracted to upper half of pleural cavity, where it is adherent by a number of bridges of fibrous tissue to costal wall. The cavity is dry. Lung is collapsed. Left lung after removal seems to contain air throughout, dependent parts are congested. Pleural surface is smooth except where the strands of fibrous tissue bands bound lung to thorax wall. No perforation apparent. The bronchi are congested. No tubercle visible or palpable.

Right lung is slightly adherent to chest wall and diaphragm about middle lobe. Lung is voluminous; contains air throughout. Dependent parts are congested. No tubercle visible.

Both layers of peritoneum smooth. Intestinal coats pale. Appendix vermiformis is swollen, firm, vessels over it are injected, it has opaque greyish-yellow color, measures about 6 cm. long, diameter uniform, about 7 to 8 mm. On cutting into it a thick yellowish pus escapes. Mesenteric

glands in the immediate neighborhood are congested and enlarged. Mesenteric and retroperitoneal glands generally not enlarged.

Pericardium contains a small amount of fluid. Layers smooth. Heart rather soft, small, and pale. Endocardium smooth, valves normal. On section muscle appears pale, greyish-pink and smooth. Coronary arteries are prominent on surface of heart; not atheromatous.

Spleen is rather small, flabby, capsule wrinkled. Surface on section, deep purple red in color, trabeculæ prominent, surface rather dry.

Liver is of moderate size, surface is smooth, somewhat pale, consistence diminished. Surface of section smooth. No tubercles visible. In substance of liver is a small anæmic area near inferior surface. No foci of necrosis visible. Gall-bladder large, contains black bile and 20 to 30 small firm, yellowish, translucent granules, the size of a coriander seed. Gall-ducts are large and patent.

Kidneys are of the same size. Rather diminished in consistence. Capsule strips off without difficulty. Surface of section; cortex markings distinct, white striæ swollen. Through cortex many opaque granules are visible after close inspection, not numerous enough to represent glomeruli, look like minute areas of fatty degeneration. Pelves of kidneys are pale, contain turbid, purulent looking material. Ureters normal. Adrenals normal. Medulla deep brown-red in color.

Bladder small, contracted, mucous membrane somewhat congested. Vagina and uterus appear normal, except that mucous membrane of cervix uteri seems striated, yellow opaque, making line of union between cervical and corporal mucous membrane very distinct. In right broad ligament, apparently attached to end of slightly elongated atrophic ovary, is a cyst the size of a small fist, thin-walled, semi-translucent and fluctuating. It lies on broad ligament and has fallopian tube extended over it. On left side tube and ovary are normal. No abnormal adhesions about uterus.

Stomach and intestines show nothing abnormal except that mucous membrane of cæcum about origin of appendix is congested. Large intestine contains semi-solid, yellow fecal contents.

Brain.—Dura is not adherent to calvarium. Several Pacchionian bodies are larger above vertex. Sub-dural space contains an excess, 150 cc., of clear straw-colored fluid. There is slight œdema of leptomeninges in sulci over vertex, more marked at base of brain. Hypophysis cerebri congested. There are no tubercles visible anywhere. Veins of pia slightly congested. Brain substance appears normal. Medulla and cord immediately below it also appear normal. On section the lateral ventricles are enlarged, distended with clear serum. Choroid plexus injected and slightly œdematous. Venules in ependyma of ventricles perhaps slightly more congested than normal. Amount of fluid in both ventricles not more than 50 cc. (not measured).

Cord.—The dura on posterior surface of cord, from 3rd to 7th dorsal vertebræ, is thickened distinctly and congested. The subdural space apparently does not contain any excess of fluid. The cord and soft meninges throughout appear normal.

This case is of particular interest. We know of no similar on record. The patient was in the ward for some weeks with symptoms suggesting meningitis. The appendicitis found at the autopsy was not recognized during life. The patient's spleen was tapped that the blood might be examined for malaria to explain the temperature of the patient and as a result pneumothorax developed. The signs were very clear. There was silence on quiet breathing, the breath sounds were distant and amphoric on deep respiration. The heart was dislocated to the right of the sternum. Six days later the breath sounds were slightly heard over the left side and a slight friction was present at the point of puncture. At the autopsy the pneumothorax of the left side was found to be pure, no fluid being present. The presence of air was proven by the water test. A striking feature was the position of the diaphragm. The lung was collapsed but contained air throughout. No perforation was found. There are two interesting points in connection with this case. It is a case of pneumothorax caused by puncture of the chest with a hypodermic needle. It seems, however, to have had no unfavorable influence on the course of the disease. Another important point was the absence of respiratory sounds on quiet respiration and their distant amphoric character on deep respiration, since the puncture of the lung doubtless closed early.

CASE 35.—*Hydropneumothorax due to puncture; recovery.*

James B., aged 26 years, white. **Med. No. 9287.**

Admitted November 29, 1898.

Discharged January 5, 1899.

CLINICAL DIAGNOSIS.—*Double acute lobar pneumonia; hydropneumothorax due to aspiration.*

COMPLAINS of pain in the left breast and general over the back.

FAMILY HISTORY good.

PAST HISTORY.—Has had rheumatism for six months, several of the joints being affected, but has not been confined to bed. Pneumonia last year; was in bed three or four weeks. His work has been hard, subjecting him to considerable exposure, but has not been severe for the past five years.

PRESENT ILLNESS.—Began November 25 with pain in his stomach, which has been getting worse all the time; headache has been constant for four days. One week ago he had nose-bleed and once since; no sweating; no chills nor chilly feelings. Cough for four days, chiefly in the morning when he rises. Expectorates little. This and yesterday morning the sputum was mixed with blood. He has a good deal of pain in the chest on

coughing. No shortness of breath. No nausea; no vomiting; no œdema, no loss of weight.

Physical Examination (Dr. Futcher).—Patient well nourished; lips and mucous membranes of good color; temperature ranges from 100.8° to 100.2°. Pulse, 104. Thorax fairly well formed. Right clavicle a little more prominent than the left; slight flattening below the right. Expansion good and equal on both sides.

Heart sounds best heard in 5th i. s. 8½ cm. from the mid-line.

Right side.—There is slight increase in the vocal fremitus below the clavicle; slight impairment of resonance above and below the clavicle as low as the third rib. Note elsewhere clear. Breath sounds a little harsh above and below the clavicle. Expiration slightly prolonged; no adventitious sounds. Breath sounds elsewhere clear.

Left side.—Percussion note is clear throughout the front; slightly impaired in the lower axillary region. Expiratory murmur slightly prolonged just below the clavicle, but not tubular. In the lower axillary region breath sounds are rather enfeebled and indistinct. Expiration slightly distant, tubular quality. In the 6th interspace outside the mammillary line a slight superficial friction rub is to be heard.

Backs.—On the right the vocal fremitus apparently normal. Percussion note practically flat below the angle of the scapula. Throughout the subscapular region there is distant tubular breathing, with very fine râles heard occasionally on inspiration. Voice sounds have a distant nasal quality. Left side.—Flatness extends as high as the middle of the scapular region. Voice sounds practically absent over the left base. In the lower interscapular region breath sounds distant but feebly tubular.

The abdomen is negative. Liver and spleen not palpable.

December 2. In the left back there is a board-like flatness and marked resistance on percussion as high as the angle of the scapula. Over the whole of the subscapular area, however, there is distant but typical tubular breathing. Vocal fremitus is absent. Right base.—Percussion note still distinctly impaired; breath sounds have still a tubular modification but not as distinctly tubular as at first. Apex beat in the 5th interspace 5 cm. from the mid-sternal line.

December 4. Owing to the height of the flatness in the left back (from the middle of the scapula down) and the increasing difficulty in respiration aspiration was performed in the 8th space just inside the angle of the scapula. About 200 cc. of serous fluid was obtained with some difficulty. Pain around the heart complained of. There is an area of superficial emphysema extending over the upper part of the right front and over the right back below the spine of the scapula.

Six hours later (Dr. Futcher). The left side of the chest is distinctly fuller than the right. There is subcutaneous emphysema throughout the whole axillary region. The percussion note throughout the whole of the left front and axilla is distinctly hyperresonant with a suggestive tympanitic quality. The breath sounds are somewhat harsh over the upper right front, indistinct over the lower left front. Over the upper right axillary

region there is typical tubular breathing. Breath sounds almost absent over the lower axillary region. Left back.—Percussion note over upper left back as low as the middle of the scapula is hyperresonant and has a somewhat tympanitic amphoric quality; modified flatness below. Breath sounds over the upper left back practically normal. Commencing just below the spine of the scapula and throughout the lower interscapular and upper subscapular regions is distinct tubular breathing. Below the breath sounds are indistinct; tubular; bronchophony is present except at the extreme base. There is no definite coin sound to be elicited. Succussion not to be made out. Respiration a trifle labored; 36 to the minute.

December 7. The coin sound was distinctly obtained. Otherwise the symptoms much the same.

December 11. Dr. Osler: Movable dulness indistinct.

December 12. Dr. McCrae: Over the left front the note on percussion in the first interspace is almost flat. Towards the outside close to the sternum it is tympanitic, as also in the second and third spaces. Just below the clavicle the breath sounds are distant and slightly tubular. Over the tympanitic area they are heard feebly; expiration is not greatly prolonged. Percussion note in the axilla has a marked amphoric quality. Breath sounds high pitched and somewhat amphoric. In the lower posterior axillary and subscapular regions the percussion note has a marked amphoric quality; apparently unchanged with the mouth open. Close to the mid-line the note has a flat, tympanitic quality. Over the amphoric area the breath sounds are distant, slightly tubular and amphoric toward the mid-line. The coin sound is not marked.

December 14. The apex beat in the 4th space 8½ cm. from the mid-line. Relative cardiac dulness extends 8½ cm. to the right from the mid-line. A tympanitic area is present to the left of the sternum extending 6¾ cm. from the mid-line in the first space and 7¼ to the left in the episternal notch. This area comes to a point joining the sternum in the 3rd interspace. All the rest of the front is absolutely flat. The absolute liver dulness begins at the upper margin of the 6th rib and extends 4 cm. below the costal border in the mammillary line. The chest was aspirated and 1300 cc. of fluid withdrawn. The tympanitic area remains the same. The flat area has changed in extent, giving high-pitched tympany with an amphoric quality in the left axilla. The point of maximum impulse of the heart has moved to the left 1½ cm.

December 19. The aspiration was again attempted, but only 100 cc. of fluid obtained.

December 22. Dr. Osler: Vocal fremitus well felt above the spine of the scapula; diminished below. A flat tympanitic note is present in the lower left back; no amphoric respiration on the left side.

December 28. The temperature has been normal for the past few days. Patient has much improved.

Chest.—Right front is normal throughout. In the left front vocal fremitus is fairly well marked throughout the front and upper axilla, just palpable in the lower axilla. The percussion note is impaired throughout

the front and axilla and is almost flat. The breath sounds are fairly well heard over the fronts, vesicular in character, no adventitious sounds. Backs.—Right back is normal throughout. In the left back vocal fremitus present throughout the whole back. The percussion note is somewhat impaired, but not as flat as it has been. Breath sounds are heard, but are somewhat muffled throughout.

Heart.—Point of maximum impulse is visible and palpable in the 4th interspace 7½ cm. to the left of the sternum from the mid-line. Sounds normal.

January 5. There is still relative dulness in the greater part of the front and back of the left chest. Vocal fremitus everywhere well felt. Breath sounds everywhere audible but feeble. The left chest expands much less than the right. Distinct visible contraction of the left side. The apex beat of the heart is present in the 4th interspace 6 cm. from the mid-line. The physical signs over the left lung are those of a thickened pleura. Patient discharged to-day.

April 16. The patient returned to the dispensary. There is dropping of the left shoulder, diminished expansion and impairment of the percussion note throughout the entire left side, especially in the lower axilla and back. Breath sounds are everywhere feeble throughout the left side, but are heard to the extreme base. The voice sounds are heard loudly in the lower left axilla and have a slightly vesicular quality. The fremitus is almost absent on the left side, but is still felt to the extreme base. During the patient's stay in the hospital the leucocytes rose but once, on December 12, to 12,000. The leucocytes during December were raised varying from 11,000 to 12,000. The temperature during the stay in the hospital was elevated, at first varying from 100° to 103°, at one time reaching 104.8°, but gradually falling to normal.

In this case of acute lobar pneumonia, which December 4 was aspirated, there is no doubt but that pneumothorax resulted. The general subcutaneous emphysema, the transformation of a flat chest to a hyperresonant one can leave little doubt on that point. Yet it is interesting that amphoric breathing and succussion were both absent while the coin sound was distinctly obtained. The opening into the lung and chest closed at once without doubt. It should be remembered that the lower lobe of the left lung was consolidated hence could not collapse. This may explain some of the abnormalities in the signs.

CASE 36.—*Pyopneumothorax due to aspiration of a pyothorax; death; autopsy.*

James L., white, aged 26. **Med. No. 13,200.**

Admitted July 26, 1901.

Died August 1, 1901.

Diagnosis.—*Pulmonary and pleural tuberculosis; pyopneumothorax.* He complains of shortness of breath.

Family History.—Good.

Past History.—Excellent. He had malaria at ten years of age. Otherwise has been a healthy man; habits good. Occupation is a glass worker.

Present Illness.—The patient had a sharp pain in the left side last Christmas Eve. This pain lasted about two weeks. After this time the patient says he began to fill up with fluid and get short of breath. He was tapped five times between the 20th of February and the 22d of May. He was in bed all of this time, during two weeks of which he was in a hospital, where he was tapped twice. He has no cough, no expectoration, no night sweats, no chills. He says he has lost 70 pounds. Had marked respiratory distress for the past two weeks.

Physical Examination (July 27, Dr. Futcher).—The patient is still rather dyspnœic and emaciated. Temperature not above 100 degrees since admission. On admission there was flatness on the left side to the second rib in the recumbent posture and to the first space in the erect posture. Vocal fremitus: Breath and voice sounds remarkably feeble over the area of dulness. The heart was pushed to the right so that the relative dulness extended to the nipple line of the right side. The patient was tapped last night; the fluid contained a great number of tubercle bacilli.

Lungs.—The percussion note is moderately resonant over the left chest. Vocal fremitus felt throughout but diminished. Breath sounds everywhere enfeebled. An occasional moist râle audible on inspiration.

Right lung.—The percussion note is a trifle impaired as low as the 3rd rib in front; above it respiration is slightly prolonged and higher pitched.

Heart.—The cardiac impulse is most distinctly seen at the right of the sternum. The sounds are heard in the 7th right interspace 6 cm. from the median line. Liver flatness extends 2½ cm. below the costal margin in the nipple line. The abdomen is distended.

July 29. During the night the patient had some dyspnœa. This morning he is sitting up in bed with considerable respiratory distress. The chest is fairly symmetrical. The left clavicle slightly more prominent than the right. Breathing chiefly abdominal. Expansion is a little better on the right than on the left side. There is no marked flattening of the left side. The intercostal spaces are better seen on the right. Vocal fremitus good over the entire right side. On the left side it is distant above the second rib but absent below in front and below the spine of the scapula behind. The percussion note over the entire right side except over the heart is resonant. The axilla relatively dull; also the back; nowhere absolute flatness.

Left side.—With the patient in the erect position note is resonant as far as the 3rd space, becoming flat over the 4th rib. The note is nowhere tympanitic. As the patient lies back the dull tympanitic quality extends from the second rib to the stomach tympany. Right side is resonant behind. Along the interscapular space the note has a tympanitic quality near the spine and becomes duller in the axilla.

Left side.—The note is impaired below the angle of the scapula.

Auscultation, right side.—Breath sounds are well heard over the upper front; rather harsh and accompanied by numerous mucous râles. Behind the breath sounds are harsh with numerous râles. Nowhere tubular.

Left side.—The breath sounds are of normal quality above, very distant, with few râles; below the third rib they become almost entirely suppressed. No metallic râles. Below the spine behind breath sounds are almost entirely absent. The coin sound is distinctly heard. The succussion splash is obtainable. The apex beat is best heard in the 4th interspace 7 cm. from the mid-line.

July 30. Dr. Futcher: The displacement of the heart to the right, notwithstanding the aspiration, the marked grade of movable dulness, the tympanitic percussion note, distinctly audible succussion and the perfectly typical coin sound leave no doubt as to the presence of pneumothorax. Breath sounds are nowhere tubular. The vocal fremitus is slightly diminished throughout. No metallic tinkling râles audible in the chest.

August 1. The patient died suddenly this morning.

The urine had a trace of albumin. The temperature was irregular, from 96° to 101.5°.

Auotpsy No. 1771.

Anatomical Diagnosis.—*Chronic pulmonary tuberculosis. Fibroid induration of right apex with miliary and conglomerate tubercles. Double chronic tuberculous pleurisy with thickening and fibrinous exudation. Left-sided pyopneumothorax. Localized pleural effusion at right base. Tuberculous pericarditis with obliteration of the pericardial cavity. Miliary tubercles in liver and spleen. Tuberculosis of prostate. Beginning tuberculous peritonitis.*

Body of a sparely nourished man, 160 cm. in length. Rigor-mortis slightly marked in the lower extremities.

After removing the skin and covering the exposed surface with water, gas was demonstrated in left pleural cavity after puncture. The cavity is found to contain, in addition to gas, about 800 cc. of turbid, dirty, dull, reddish fluid. The lung is displaced to the posterior and inner part of cavity, occupying only a very small space. The parietal pleura is enormously thickened and stripped off from the chest wall is found to have an almost uniform thickness of 7 mm. The inner surface is of a greyish yellow color and presents superficial irregularities, though relatively smooth. On section the greater part of the thickened pleura consists of greyish yellow opaque material, apparently fibrinous exudate, below which there is a thin layer, 1 mm. in thickness, of greyish-white translucent tissue. The external surface of the parietal pleura shows the markings of the ribs. Minute vessels are conspicuous. The pleura covering the lung has the same character, though the fibrinous exudate covering the surface is slightly thinner, from 2 to 4 mm. Between the inner surface of the compressed lung and adjacent mediastinum there is a considerable accumulation of exudate, which is quite soft and friable and resembles caseation. There is no per-

foration of visceral pleura at any point. The right pleural cavity is almost wholly obliterated by adhesions, binding the inner and external surfaces of the lung to parietal pleura. At the base of the lung, however, is a localized pleural cavity containing probably 250 cc. of clear, straw-colored fluid. The pleura lining this cavity, over the diaphragm and base of lung, is enormously thickened by an exudate, similar to that already described.

Left lung is of very small size, measuring 16 x 5.5 x 7 cm. The tissue is tough and leathery. On section it is of a uniform dull, deep red color, mottled with coal pigmentation. At the extreme apex is an area about 2.5 cm. across, thickly studded with grey tubercles, usually 1.5 mm. in diameter, between which is a small abount of fibrous tissue. Crepitation can hardly be felt, yet lung floats in water. Bronchi are not injected. Several of the bronchi of the lower lobe, joining to form one of the larger branches, are studded with irregular, grey-yellow erosions, between which the mucosa is slightly injected. No perforation into pleural cavity could be traced.

Right lung is voluminous, pinkish-grey in color, moist, crepitant throughout, though thickly studded with discrete confluent tubercles, greyish-white, often with opaque center. The uppermost 3 cm. of the apex is occupied by a very firm, fibrous, deeply pigmented tissue, which is completely airless. At its periphery are confluent caseous tubercles, while within it are a number of opaque white areas, about 3 mm. across and firmly calcified. The bronchi are slightly injected.

This patient with tuberculous pleurisy was evidently admitted with pneumothorax, since the heart on admission was chiefly to the right of the sternum. But the physical signs were most manifest July 29. We fear this case was due to aspiration. It is of interest that here, where there was no trace of a fistula found, the amphoric breathing was absent, and no metallic tinkling present. The lung was very much compressed, the pleura much thickened, hence hyperresonance was not a feature in the case and succussion and the coin sound were very distinct.

CASE 37.—*Hydropneumothorax due to the aspiration of a hydrothorax; recovery.*

Lewis E. L., white, 25 years old. **Med. No. 13,604.**

Admitted October 19, 1901.

Discharged December 9, 1901.

CLINICAL HISTORY.—*Tuberculosis, pleural and peritoneal; pneumothorax.*

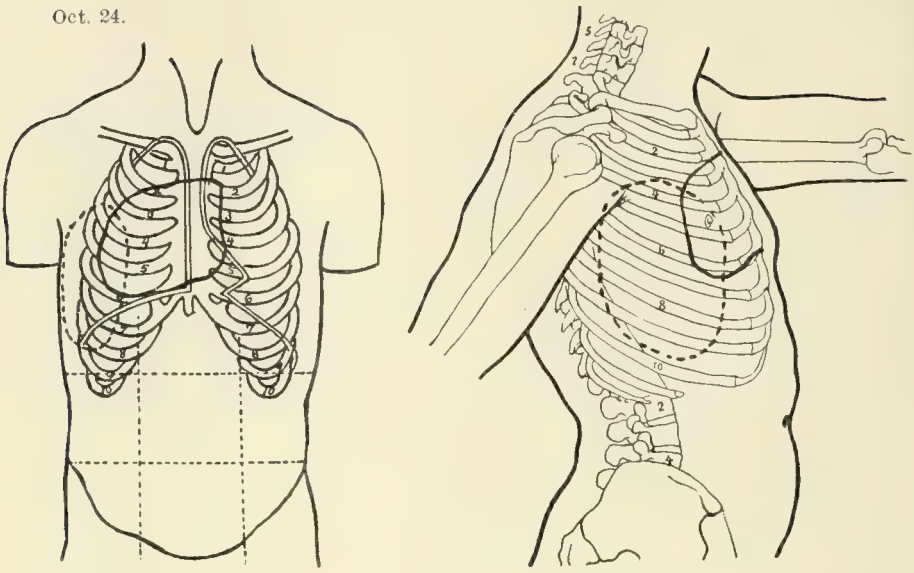
The patient COMPLAINED of pleurisy with effusion on the right side and ascites.

FAMILY HISTORY negative.

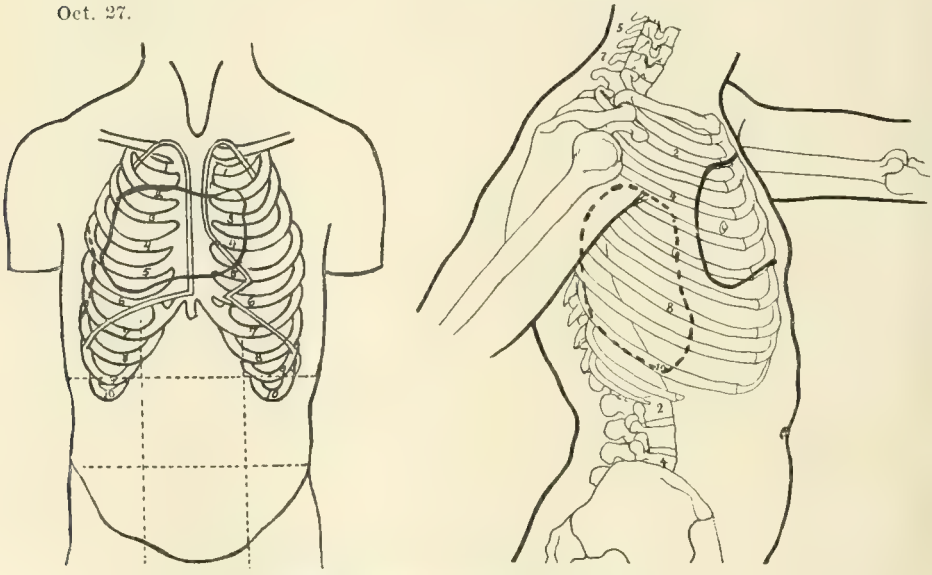
PAST HISTORY.—Whooping-cough is the only children's disease he has had. No pneumonia or pleurisy. He had malaria when 23 years of age

L. E. L.

Oct. 24.



Oct. 27.



BLACK.—Area of tympany in patient on back.
DOTTED.—Area of tympany in patient on left side.

without shaking chills and without a subsequent attack. Jaundice, one or two slight attacks a year for the past five years or so. Marked attack last March. He has had nasal catarrh for six or seven years; denies all other diseases.

Present illness began March 1 after exposure to drafts for about two weeks. The onset was with headache, slight cough. The diagnosis which he made was grippe. After two weeks he stopped work and consulted a doctor. Six days later he came to Baltimore. The physician who treated him here said his right lung was consolidated and that numerous tubercle bacilli were present in his sputum. Four weeks later an effusion on the right side, which was tapped, developed. The patient had no pain except a severe temporary pain in the right shoulder. He had sweats occurring irregularly. In the sixth week the lungs began to clear, with cough and expectoration of a white sputum. Patient was in bed nine weeks, after two weeks of convalescence went to the country, where he remained until August 16, gaining five pounds. He felt well, but had not regained his former strength when he returned to work. Until October 1 he felt quite well and then said was compelled to drink bad well-water, which did not agree with him. He lost his appetite and developed diarrhœa. A few days later his abdomen began to swell and became sore. He worked until October 18, when he came to the hospital. For about a week he noticed he was getting short of breath, but his right side gave him no pain and he did not know that the effusion existed in that pleural cavity. For a week the patient has had pain in his stomach, which felt full and seemed to have some weight upon it. No digestive disturbances.

Physical Examination.—The patient is emaciated; not anæmic.

Thorax.—The clavicles are prominent. The right side is fuller than the left. The intercostal spaces obliterated and limited on respiration. On percussion the right side is dull at the upper border of the second rib and flat at the third rib in the parasternal line. In the back it is absolutely flat below the angle of the scapula. On the left side the note has a low, muffled, tympanitic quality, but is quite resonant. Tactile fremitus is completely absent on the right side; present on the left. Over the right apex the breath sounds are quite feeble, accompanied by a few medium râles.

Over the remainder of the right side the breath sounds are practically absent, especially below the 3rd rib. On the left side the breath sounds are everywhere harsh and expiration prolonged and bronchial in character.

Heart.—Point of maximum impulse is in the 5th space 15 cm. from the median line. No abnormalities in sound. The pulse is 100; good volume. The abdomen is quite full, respiratory movements slight. Costal grooves obliterated; umbilicus prominent. The flanks are full and protrude. On percussion there is definite movable dulness in both flanks. Fluctuation readily obtained. Nothing is made out on palpation. There is no œdema of the lungs. Marked myoidema.

October 20. The pleura was tapped; 600 cc. removed.

On the 21st the right pleura was tapped again and 1500 cc. removed.

Oct. 22. No marked dyspnœa. Over the right supra- and infraclavicular regions the note is resonant, but compared with the left side, impaired. Below the 2nd rib the note is dull. Between the 3rd and 6th ribs it has a distinct amphoric quality, marked bell tympany. In the axilla the note is flat as high as the 3rd space. On auscultation above the breath sounds are well heard and of normal quality. Below there is silence, save for an occasional amphoric râle (the patient recumbent). Behind is resonance above with normal breath sounds; below is flatness and silence. With the patient erect the coin sound is well heard, but the succussion splash cannot be obtained. The point of maximum cardiac impulse is in the 5th i. s., 12.5 cm. from the mid-line. (The most of this note is omitted, since the following is more complete.)

October 24. Dr. McCrae: The patient looks better. There is no dyspnœa. Respiration is good; well marked on the right, but more on the left. The note over the right clavicle is much impaired. In the 2nd, 3rd, 4th and 5th right interspaces over the whole body of the sternum and in the 2nd, 3rd, 4th and 5th left interspaces for a distance of about 4 cm. from the median line there is most intense tympany which extends to the right to about the anterior axillary line. That is in the recumbent posture. When the patient sits up it completely disappears and is replaced by flatness. When the patient lies on the right side the tympany is still present, but does not extend as far as the right mammillary line. When the patient lies on the left side the position of the tympany changes markedly and shifts to the uppermost point, that is, the right axilla. It extends anteriorly only a little in front of the nipple and back almost to the posterior axillary line. When the patient turns almost flat on his face there is practically no change from that observed when he is on his left side. The percussion note was the same pitch with the mouth open and shut. The coin sound not obtained. Succussion was obtained with the patient lying flat but not when sitting up. On auscultation there is distant breathing heard which has not an amphoric quality. No especial alteration in the voice sounds. Vocal fremitus quite readily felt over the whole area of tympany in front, but not felt through the axilla either with the patient on the back or on the left side.

October 25. Dr. Osler: Marked suppression of the breath sounds; no amphoric quality on cough; distinct succussion.

October 27. Dr. McCrae: Tympanitic area with the patient on the back has extended considerably. It goes just as far to the left of the sternum, but now to the right middle axillary line. There is a most intense tympany in the 4th and 5th right interspaces just outside the nipple line, yet the tympany completely disappears when the patient sits up.

October 28. Dr. Osler: Marked coin sound; marked amphoric quality on coughing.

October 29. The girth of the abdomen has diminished about 5 cm.

November 4. The patient was aspirated to-day, the needle inserted in the 8th interspace of the scapular line. Following the insertion of the

needle and opening of the valve into the aspirating bottle there was a rush of air which caused the connecting tube to vibrate. Patient also said that he could feel his chest grow easier and seem less full. After a short interval the fluid also began to appear and about 600 cc. of a cloudy hæmorrhagic fluid were withdrawn. Following this the chest became hyperresonant in front, the tympany disappeared. The breath sounds and the fremitus are easily discernible. In the mid-axilla there is still a small area of tympany, which, however, did not shift with the patient's position. Below the angle of the scapula behind is flatness with suppression of breath sounds. Distinct succussion was obtained; no coin sound. No tubercle bacilli could be found in the exudate.

November 20. The patient was aspirated this morning and 850 cc. of blood-stained fluid withdrawn. Following the aspiration the movable dulness much decreased; coin sound absent; only slight succussion. The tympany is absent but the note around the nipple has a muffled, tympanitic quality.

November 21. With the patient sitting up there is resonance in the front as low as the 4th rib. There is no marked tympanitic quality. When the patient lies down there is a muffled tympany over the whole right front and the upper axilla; flatness over the lower axilla and the lower back to the angle of the scapula. Breath sounds are well heard over the right front. Expirations are prolonged and rather harsh. The breath sounds are suppressed over the area of flatness. The apex is apparent in the 6th interspace 9½ cm. from the midsternal line.

November 30. The patient was aspirated again and 500 cc. removed. There is still marked movable dulness and succussion; coin sound not obtained; no tympany over the chest.

December 9. The patient has been gaining in weight and strength rapidly and was discharged to-day. During his stay in the hospital his temperature was irregular, from 97° to 101.5°, practically normal after November 18. "This pneumothorax was accompanied by curious signs. It may have been due to the aspiration." This is the comment of the medical resident as the patient left the hospital.

The urine was negative; no albumin casts or Diazo test.

Second admission, March 10, 1902. **Med. No. 14,122.**

Discharged March 15, 1902.

Clinical Diagnosis.—*Pleurisy with effusion; tuberculous pneumothorax.*

February 12. The patient was aspirated at the Johns Hopkins Hospital and 1500 cc. of turbid fluid were obtained from the right chest.

Present Illness.—A week ago the patient noticed shortness of breath on exertion. Also bulging on the right side. He had soreness in the stomach above the umbilicus since December 1, when he last left his bed. The patient also notices that the heart is rapid. He does not complain of any pain or discomfort. Weight remains about the same. General condition is good.

Physical Examination.—The patient looks well nourished; color good.

There is no dyspnœa, although some on exertion; no cyanosis. The right side of the chest is flatter than the left. The right clavicle is more prominent and expansion is markedly less on that side. The percussion note over the left front and axilla is rather hyperresonant; impaired over the right apex below. There is flatness over the lower sternum. There is some tympany which extends over the greater part of the body of the sternum and shades off into hyperresonance. It extends somewhat to the left; does not change with change of position. There is flatness throughout the right back except at the upper portion. The left back is clear. The breath sounds are harsh but clear on the left. They are heard feebly over the right apex; otherwise they are absent throughout the right side.

Vocal fremitus is absent over the greater part of the right side. The breath sounds are faintly heard from the upper portion of the tympanitic area in front. No especial quality.

Heart.—The apex beat is in the 5th interspace 13 cm. from the mid-line. The area of dulness is practically obliterated. The sounds are loud and booming but clear. The abdomen is natural, apparently no effusion. Neither the coin sound nor succussion was obtained.

March 11. The patient was aspirated and 1150 cc. of turbid, greenish-yellow fluid were removed. Now there is definitely no expansion on the right side. There is resonance to the 6th rib in the anterior axillary line, below which flatness, which, however, in the 6th and 7th spaces has the quality of flat tympany. The area of tympany noted this morning has moved to the right about 10 cm. and now has the right nipple for its center. Is approximately the same size when the patient sits up. When the patient sits up the area of tympany completely disappears and is replaced by dulness below and somewhat dull tympany above. Over this area of tympany there is unusual vocal fremitus when the patient lies flat. It is much diminished when he sits up. Breath sounds are rather better heard; no striking change, however; no amphoric quality. When the patient turns on his left side the area is shifted into the axilla as before. Definite succussion is obtained.

Heart.—The impulse is much less marked. The apex is difficult to determine. The sounds are best heard 3 cm. to the right of the point of maximum impulse this morning. On examination of the pleural fluid tubercle bacilli were found.

March 14. The patient was aspirated and 900 cc. of fluid obtained. After aspiration there is still impairment of expansion on the right side. Below the 6th rib in front is flatness. Just below and outside the nipple is an area of tympany 4½ cm. wide and 6 cm. in vertical direction.

The heart sounds are best heard in the 5th interspace 9 cm. from the midsternal line.

March 15. The patient discharged in good condition.

The urine was negative; no albumin; no casts.

Temperature practically normal.

In this case the patient with pleurisy with effusion was tapped on October 21 and 1500 cc. aspirated. The presence of pneumothorax

was undeniable, since unfortunately the aspirating bottle was attached to the wrong valve of the suction pump, and it was the dyspnœa and inflation of the side that called attention to the mistake. The following day were noted the unmistakable signs of pneumothorax: an area of intense tympany which seeks the highest point in the chest in all positions. It is evident that the lung prevents it rising to the top when the patient lies on his face, since the signs (October 22) show it was situated there. The coin sound was heard with the patient erect. The succussion splash could be distinctly elicited with the patient lying flat but not when sitting up. On auscultation is heard distant breathing which has not an amphoric quality. Later Dr. Osler noted: marked suppression of the breath sounds, distinct succussion, marked coin sound, marked amphoric quality on coughing (not present four days before). November 4 on aspiration considerable air left the chest, following which the chest instead of tympanitic became hyperresonant.

This patient was aspirated several times more. It is interesting that although the air was certainly present the coin sounds or the succussion splash could not often be obtained (see note of November 30).

The patient left the hospital but returned in three months with much the same condition. There was a tympanitic area under the sternum, but neither the coin sound nor the succussion splash were obtained until he was again tapped, then the area of tympany changed its position and disappears when the patient sits up. The breath sounds have no amphoric quality, yet the succussion is now easily obtained.

The points to which especial attention are invited are (1) the small amount of air which sought the highest point in the chest; (2) the fact that succussion was not always obtained, or only when the patient was on his back; (3) the absence frequently of a coin sound; (4) the absence of amphoric respiration.

CASE 38.—*Pneumothorax due to aspiration of a hydrothorax; recovery.*

Eugene C., colored, 10 years old. **Med. No. 14,279.**

Admitted April 17, 1902.

Discharged May 5, 1902.

DIAGNOSIS.—*Pleurisy with effusion; pneumothorax. The patient complains of cough and pain in the right side.*

FAMILY HISTORY is good. No tuberculosis.

Past History.—The patient has had no pneumonia or pleurisy. He has had occasional night sweats, but not regularly. He has never had cough or pain in the chest. The appetite has been poor for some months past; habits have been good.

Present illness began April 10, 1902, with sore throat on waking up. That day he refused food; was up and about, but tired and weak. That night the sore throat was worse. There was no pain in the side and no cough this day. The next day the throat seemed better, but that evening he began to cough for the first time, the cough keeping him awake; no expectoration; no pain in the side. Two days later the cough increased a good deal and continued for three days more. At night he had a definite chill before going to bed, followed by a sweat. This day also he had a sticking pain in the right side on walking around and an occasional one in the left side. He has had fever and three night sweats since the onset. There has been no sputum. The patient has not been confined to bed.

Physical Examination.—The boy is well nourished. He has no respiratory distress.

Thorax is symmetrical, but expansion is distinctly less over the entire right side. The intercostal spaces do not bulge, although they are fuller. Fremitus diminished over the lower right back. On auscultation there is marked tubular breathing, especially well heard over the front and in the axilla. The sounds are slightly diminished over the right lower back, where distant tubular breathing is heard.

Heart.—Point of maximum impulse is in the 6th space 7½ cm. from the mid-line. The sounds are clear.

Pulse 128. Abdomen natural. The glands are generally palpable.

April 18. Dr. Osler: Expansion less on the right side. Respiration is harsh. Apex beat outside the nipple line; flatness extends over all the right front and the whole of the right back. It is singularly flat at the apex. Vocal fremitus is absent at the right base. Marked diminution at the right upper lobe. On the left side the breath sounds are loud, full and clear. On the right side they are intense, distinctly tubular. At the extreme right base there is distinct tubular breathing. The sounds are everywhere dry. The patient was aspirated and 500 cc. of a thick, bloody fluid obtained, which coagulated at once. The child struggled considerably, and following the withdrawal of the needle there was slight subcutaneous emphysema at the point of puncture. The note now is resonant over the front with movable dulness at the 5th rib. Flatness below the angle of the scapula. Tactile fremitus is well heard over the front; diminished over the right back. Breath sounds over the right side have increased in intensity but still retain their tubular modification. The note over the front has a definite tympanitic quality. No tubercle bacilli were found in the exudate.

April 19. No dyspnœa; no cyanosis. There is some bulging on the right side where movement is limited. Cardiac impulse is in the 5th space 7¾ cm. from the mid-line. Expansion is not completely absent on the

right side. Above the right clavicle the note is almost flat. It is tympanitic down the right front to the 6th rib, and to the mid-axillary line. When the patient sits up there is almost normal resonance in the upper interscapular region. Dulness from this point to the angle of the scapula, below which there is an area of well-marked tympany almost circular and about 7 cm. in diameter. When the patient sits up there is fairly good resonance above the clavicle. At the first right interspace there is good resonance, at the second it is impaired; the 3rd, 4th and 5th are tympanitic (note the change from the horizontal position). When the patient turns on his face there is still tympany in the area noted before but fairly good resonance in the back. The vocal fremitus is felt down to the base on the right side; well marked subcutaneous emphysema in the right axilla. On auscultation everywhere over the front the breath sounds are loud, harsh and blowing, accompanied by numerous râles. It is slightly more tubular throughout the axilla. It has a hollow amphoric quality at the angle of the scapula. Over the tympanitic area it is distant and blowing with moist, crackling râles; slight suggestion of the coin sound; no succussion made out. Left side normal.

April 21. Chest aspirated again; 100 cc. of clear, straw-colored fluid withdrawn. No more fluid could be obtained. The signs over the chest remain about the same.

May 2. The note over the upper right chest is changed in character. It has lost its tympanitic quality and still remains hyperresonant. It is impaired in the lower axilla and back. There is no movable dulness. The tactile fremitus is exaggerated over the right front; is well felt over the right back. The breath sounds are rough over the lower back and are accompanied by fairly numerous coarse mucous râles.

May 6. The patient was discharged to-day practically well, there being no signs of effusion and few râles are still audible over the right lower front. The urine was negative. Temperature elevated between 100 and 103 until April 23, after which time it fell to normal, reaching there about the 27th.

Note on dismissal.—"This was an interesting case. The pneumothorax was probably produced during tapping on April 18. It was not accompanied by any bad results."

We have here a case of pleurisy with effusion on the right side. Following aspiration, during which the child struggled, there developed subcutaneous emphysema, resonance with a tympanitic quality, marked movable dulness and the breath sounds became loud, harsh and blowing; yet the coin sound was only suggestive and the succussion splash was not obtained.

CASE 39.—*Hydropneumothorax, due to aspiration of a hydrothorax of the left side; recovery.*

Zadok H. D., 17 years old, white. **Med. No. 14,370.**

Admitted May 14, 1902.

Discharged July 1, 1902.

Clinical Diagnosis.—*Tuberculosis of the pleura, pericardium and peritoneum. Pulmonary tuberculosis. Pneumothorax, due to tapping.*

Complaint.—Cough.

Family History.—One sister died of consumption.

Past History.—Has had most of the diseases of childhood. He has never had malaria, pneumonia, pleurisy nor rheumatism. He has never had cough or night sweats. His gastro-intestinal tract has never troubled him. His habits have been good.

Present Illness.—This began in January, 1902, the patient feeling dull and tired. This feeling of lassitude lasted until March, when the patient commenced to have a cough. This has been worse at night, so severe that he must sit up in bed to ease it. His sputum is most abundant in the morning, is whitish with hard pieces in it. In March he expectorated a mouthful of blood, and has had night sweats and a pain in the side since that time.

About three weeks ago there was swelling just below the ribs in the median line. He became short of breath at this time. Five days ago began swelling about the ankles.

No headache, no abdominal symptoms, no increase in micturition.

Physical Examination.—The patient is well nourished. He is sitting up in bed with moderate dyspnœa. Pulse 152 to the minute, regular, of low tension, the artery's wall can be felt.

The thorax is fairly well formed. The lower left axillary region expands more than on the right, and the intercostal spaces are not so well seen on the left side. Litten's sign is not present.

Right lung.—Vocal fremitus is well felt throughout. There is movable dulness reaching the 5th i. s. in the erect posture in the mammary line, the upper border of the 7th rib in the recumbent posture. Posteriorly the flatness reaches the angle of the scapula. At the apex expiration is a trifle prolonged with an occasional râle. In the 4th and 5th i. s. of the anterior axillary line is a distinct friction rub, also heard at the angle of the scapula. Below, the breath sounds are diminished and accompanied by râles.

Left lung.—Vocal fremitus is increased in the infraclavicular, diminished in the axillary and absent in the subscapular region. In the 1st and 2nd interspace is definite Skodaic resonance. There is movable dulness of one interspace, reaching the 3rd rib in the erect posture. In the apex, 1st and 2nd interspaces, a distinct pleuritic friction rub is heard, with a definite cardio-respiratory rhythm. Throughout the axillary region the breath sounds are enfeebled, but on inspiration are heard quite a number of superficial crepitant râles. In the axillary region ægophony is present. Over the upper back the breath sounds are well heard and are accompanied by medium moist râles. Below, they are distant with a distant tubular quality.

Heart.—Apex beat neither seen nor felt. The sounds are best heard in the 4th interspace, 3.5 cm. from the mid-line. The relative cardiac dulness begins at the upper border of the third rib at the left sternal border and extends to a point in the 4th interspace 7.5 cm. from the sternal margin, where it merges into the pleural flatness. Both sounds are well heard at the apex. In the 3rd space is a definite pleuropericardial rub The second pulmonic is accentuated. Along the right sternal border is a to-and-fro friction rub.

The hepatic flatness extends 6.5 cm. below the costal margin in the mammary line. There is some fulness in the epigastrium. The abdomen is full with suggestive bulging in the flanks but no movable dulness determined.

May 16. The left pleural cavity was tapped in the 7th interspace, 1000 cc. of slightly turbid fluid removed. Subcutaneous emphysema developed about the puncture. Near the angle of the scapula is a tympanitic area where whispered pectoriloquy is heard. The left front is resonant, the base behind gives a tone of impaired resonance. The breath sounds are heard to the base with a friction rub and fine crackling râles. There was no coughing until after the needle was removed. The left side moves less than the right.

May 17. The heart's position has not changed. The dulness is exceedingly movable the fluid taking the lowest position whatever the position. Vocal fremitus is present except at the base. The front is tympanitic on percussion. Over the body of the heart a suggestive to-and-fro friction is heard.

May 19. The left side moves very little; there is still fulness of the intercostal spaces. The percussion note is resonant in the upper left front. Good full resonance over both the backs. The breath sounds are not nearly so distinct as yesterday (when they were distant with tubular quality), in fact, are almost inaudible over the left chest. The voice sounds have a nasal quality. There is no distinct apex beat, although there is a diffuse impulse over the 3rd, 4th and 5th left interspaces. No impulse is felt. There is resonance over the cardiac area down to the 4th rib. Nowhere is absolute dulness. To the right, opposite the 4th rib, dulness extends 4 cm. There is resonance throughout over the sternum. The heart sounds are fairly well heard, the first is like the second sound. There is no pericardial rub.

May. 19. Repeated examinations of sputum have shown no tubercle bacilli. The same is true of the pleural fluid.

May 24. The heart now occupies practically the normal position. There is still a large area of movable dulness on the left side, ægophony, suppressed breath sounds and still distinct splashing.

May 25. Tubercle bacilli were found to-day in the sputum.

May 29. Marked pulsus paradoxus is present. The heart sounds are still decidedly feeble and have a suggestive embryocardial quality. Down both margins of the sternum is a friction rub. In erect posture flatness is at the level of the third rib in the mammary line, while in the recumbent

posture the whole of the front and axillary region becomes of a modified tympanitic quality. No definite coin sound is heard.

June 17. For the past few days the abdomen and legs have been swollen; there is some œdema of the scrotum. The abdomen is full, tense with movable dulness in the flanks. The patient's general condition is not so good. The pulse is distinctly weaker than it has been and is markedly paradoxical.

June 20. Expansion seems about equal on both sides. There is impaired resonance over both backs and axillæ.

June 28. Patient has had some difficulty in swallowing. There is a wide area of visible diffuse pulsation over the whole front of the thorax. The apex beat, determined with the stethoscope, is in the 5th interspace 10 cm. from the mid-line. The cardiac dulness begins at the third rib. Opposite the 4th rib it extends 2.5 cm. to the right and 7.5 cm. to the left. Over the body of the heart is an interesting murmur, probably pleuropericardial, not constant but occasionally at the height of inspiration. It is curiously superficial and of a squeaking quality. It was not heard during deep inspiration.

July 1. The patient was discharged much improved.

The urine at no time during the stay showed albumin or casts. The temperature during the whole time was elevated, remarkably constant, never rising above 101°.

The note made by Dr. McCrae in commenting on the case was as follows: "An interesting case. On admission he had fluid in the left pleural cavity. After the tapping the signs of pneumothorax were noted. They were more probably brought on by the aspiration than present from the onset and overlooked. He was none the worse for the pneumothorax. There seemed to be little doubt of pericardial involvement. The signs of pneumothorax were very marked."

This is therefore a case of pulmonary tuberculosis of the serous cavities and hydrothorax of such grade that tapping was necessary. The chest was aspirated and as a result well-marked pneumothorax resulted, as shown by the subcutaneous emphysema (the punctures in these cases are always closed at once with collodion, hence the air must have come from within the chest), succussion splash, the extreme movable dulness, the resonance of a slightly distended side with obliteration of the area of cardiac dulness. It is interesting that in this case the respiratory sounds were distant and at times inaudible, for of the presence of a pulmonary fistula there is no evidence.

This series of ten cases is without doubt the largest reported. It will be seen that several of the possibilities are here realized. In one (case 37) the air was pumped into the chest, in one it was

allowed to enter through the unguarded needle (case 33), in another (case 34) the lung was pricked by a hypodermic needle. What the cause of the others was one cannot say. Possibly adhesions had formed which stretched by the expansion of the lung tore the pleura, possibly superficial cavities or emphysematous bullæ were ruptured by the by no means small negative tension developed by a good aspirating pump in the hands of a willing and strong assistant. Of one thing we are certain, that judging from these cases it is not an accident to be disregarded or made light of. The absorption of air was always slow, and in case 34 in which the pleural cavity was presumably normal, at autopsy twenty-four days later, the lung was bound down by adhesions and had not expanded at all.

It will be noted that large amounts of fluid had been removed, and the moral is to remove less fluid and not suck it out with any violent negative pressure.

Of our 10 cases 5 died. How much weight the air had in this fatal determination we do not attempt to decide.

Foreign bodies in the lungs.—Such cases are very rare. Cayley[174] thinks that in his case the fish bone pierced the lung rather than the œsophagus. The best case is that of Zuppinger,[253] in which a small ear of corn was found in the lung in a child into whose mouth it had probably fallen from the threadbare mattress with which the child was covered, and was aspirated into the lung.

Pneumonia.—This was early considered a cause of pneumothorax. It was not until Skoda had emphasized the presence of the hyperresonant note, suggesting air which is present in pneumonia and in pleurisy, over the not-yet involved lung that these cases were recognized as not pneumothorax. The case of Graves[66] is best considered one of these mistaken diagnoses. Rumpf[90] reports one case of pneumonia followed by bronchiectasis, resulting in gangrene and pneumothorax. Monneret[104] reports a case which may belong here, of pneumonia followed by gangrene and death in less than 24 hours. Taylor[182] mentions a case. The case of Paviot[258] is one of particular interest since so far as we know it is unique. It was pneumothorax, which he ascribed to chronic ulcerative pneumonia.

Typhoid Fever.—The number of cases of pneumothorax developing in typhoid fever was found unexpectedly large. There would seem to be, according to Mahomed,[188] a condition of the lungs due to typhoid fever which renders the danger of pneumothorax imminent. The first case reported is that of Marechal.[57] Cayley[196] reported one case in which it developed on the 25th day. He considered it a not infrequent complication. Some, he says, are due to gangrene or thrombus from the right heart from systole and suggests that many cases have been reported.

These, however, we did not find. White [270] later reports two more cases, both due to strain at stool. We would call attention to Case 27.

Acute Articular Rheumatism.—One case of this disease accompanied by pneumothorax is reported by Glaeser.[241]

Diphtheria.—The first notice found of these cases is given by Cnopf,[252] who reported four cases and quoted two others. He considers, therefore, diphtheria a well established cause of pneumothorax. The case of Villy [331] is ascribed to whooping-cough, but it was during convalescence from diphtheria.

Hydatids of the Lung.—Williams' [55] case is possibly an early example. Here three hydatids were present in the lower left lobe but the opening into the bronchus was not discovered. Saussier [78] mentions three other cases, in one of which 500 hydatids were expectorated within two days. Jamieson,[249] without reporting any case, mentions this as a not infrequent cause of pneumothorax in Australia.

Whooping-cough one would expect a frequent cause since the expiratory efforts are so violent. There are, however, very few cases on record. Stokes [70] seems to recognize the fact that this is considered a common cause, but he calls the attention of those reporting such cases to the fact that many of these cases are complicated by tuberculosis. Villy's case [331] is ascribed to this cause, but has been mentioned under diphtheria.

DISEASES OF THE PLEURA.

Serous Pleurisy.—Bayle was the first to claim this as a cause and illustrates it by a case. His explanation is one which we shall speak of later, the absorption of a fluid and its place taken by an aeriform fluid. This form is similar to that which Williams speaks of as a "pneumothorax of necessity." After the relation between gases and liquids had been established (Dalton) this group of cases received a new significance and the gas was supposed to be due to transformation of the liquid. In so many of these cases, however, aspiration had been performed that we will include a further discussion of the group under that heading.

Rupture of a pleural effusion through the lungs.—Thompson [142] divided all cases of pneumothorax into centripetal, of which this group is an illustration, and centrifugal, of which cases rupture of the lung is an illustration. Forbes [64] is the first to be found to add this cause to the ætiological catalogue, and bases the classification on Archer's and Hawthorne's cases. Weil reported cases due to this cause. Benjamin Bell,[54] however, mentioned as a possible cause the erosion of the surface of the lung by contact with purulent matter. He may, therefore, have seen an illustration of this group. Stokes' [70] case is interesting since the man by almost standing on his head was able to empty his chest through the mouth. Grisolle (Gérard [127a]) called attention to the fact that in a rupture of an empyema through the lung several days usually pass before the pneumothorax is discovered. In Thompson's case [143] the patient was practically drowned in the pus during its evacuation, and the same is true of

our Case 41. The question of the expectoration of the pleural exudate following aspiration is to be considered. Roy [337] considered it present in his case, but Gaillard explained Roy's case as one of albuminous expectoration.

CASE 40.—*Pyopneumothorax following a perforating empyema through the lung; operation; recurrence of empyema perforating through the lung; gangrene of the lung; operation; recovery.*

(This case was reported by Dr. Osler in the Johns Hopkins Hospital Bulletin, Vol. III, page 119.)

Theodore R., white, 19 years old, can-maker. **Med. No. 2450.**

Admitted October 7, 1892.

Transferred to the surgical side October 20.

Clinical Diagnosis.—*Pyopneumothorax.*

Complains of "cough and sometimes profuse expectoration."

Family History.—Good.

Past History.—As a boy he was well. When twelve years old he "had malarial fever, tertian type, off and on" for three years. For one year has been unable to work owing to this.

Present Illness.—Began about one year ago. He worked in a packing house where he must do heavy lifting and was exposed to draughts. The onset was gradual with tired feelings, pain in the bones, cough and expectoration. He was seven weeks ill in bed with "pneumonia." He got gradually better and "went out doors too soon, catching a fresh cold." The cough increased, was worse at night; expectoration profuse, even a cupful at once; night sweats nearly every night. During the first seven weeks he was short of breath and must lie on his right side, which is true now or cough troubles him.

About six months ago the patient came to the dispensary of the Johns Hopkins Hospital. It was then noted that the right side was more prominent than the left, and the motion on that side limited. The percussion note was somewhat higher pitched at the right apex than the left, but there was flatness from the 4th rib in front and the angle of the scapula behind, down, with absent tactile fremitus, feeble and distant breath sounds. The heart was not displaced. Exploratory puncture showed no fluid.

Physical Examination.—October 10, 1892. The patient is well nourished, good color. The fingers show beginning clubbing.

The chest is asymmetrical, the right lower zone being distinctly more prominent. The right clavicle is more prominent. This side moves very little on respiration.

Heart.—Maximum cardiac impulse in 4th and 5th interspaces at or inside the nipple line.

Palpation.—The spaces on the right are harder to feel.

The percussion note on the right side is slightly higher pitched in the superior portion than on the left, but the difference would scarcely attract notice. Nowhere is it tympanitic. Below the 3rd l. s. it is flat (in the erect posture). Tactile fremitus is well felt over the whole right front. It is absent at the base behind.

The breath sounds on the right side at the apex are distant, expiration is prolonged. On deep inspiration, below the 2nd rib, the sounds are distinctly amphoric and coughing produces loud metallic râles; these sounds are heard all over the mammary region, and in the axilla. On the 4th and 5th ribs, parasternal line, inspiration and expiration are peculiarly dry and seem close to the ear. In the upper back the breath sounds are distant and feeble, not amphoric but muffled and without râles, nor can any metallic phenomena be obtained.

The coin sound is obtained.

The Hippocratic succussion splash is heard.

The sputum was many times examined for tubercle bacilli, but at no time were any obtained.

The urine contained a faint cloud of albumin but the Diazo test was not obtained.

On October 20 he was transferred to the surgical side for operation.

Transferred to surgical side October 20, 1892. **Surg. No. 1879.**

Discharged December 9, 1892.

October 20. Operation, Dr. Halsted.

A long curved incision was made on the right side of the chest and extensive flaps dissected, uncovering the ribs external to the nipple and in the axilla. Portions of several ribs were excised and the pleura opened. The pleural cavity contained pus and gas. The cavity was drained and loosely closed. In the fluid, which was hæmorrhagic and purulent, no tubercle bacilli were found. Cultures gave pure culture of Staphylococcus pyogenes aureus.

The note made by Dr. Osler just before dismissal is as follows:

The patient goes out today perfectly well. As he stands in front there is a slight flattening of the right front but the shoulders are of nearly the same height. On deep respiration the left expands more than the right, although there is distinct expansion on this side. Resonance on the left side is full and clear; on the right front there is good resonance down to the fifth rib. In the back the contraction of the right side of the chest is much more marked and there is slight curvature to the right in the dorsal region. There is resonance throughout the right back, although it is markedly dull below; the vocal fremitus is present though diminished throughout this back. The respiratory murmur is full and clear through the left back. Throughout the right front the respiratory murmur is enfeebled but present, of a normal character and unaccompanied by adventitious sounds. Below the wound in the axilla no respiratory murmur is heard. An enfeebled respiratory murmur is heard throughout the right back; no adventitious sounds are heard.

Discharged December 9, 1892.

Second admission February 28, 1895.

Discharged March 8, 1895.

Surg. No. 3937.

The wound of the operation closed ten days after the operation. Since then he has had a cough regularly every month and would expectorate much purulent material of bad taste and odor. This cough has increased in severity and comes in spells of two or three days' duration, and two hours a day. At the end of coughing he expectorates blood.

PHYSICAL EXAMINATION.—Heart: The apex beat is in the normal situation.

Lungs.—Left, normal throughout. Right, expansion diminished. The side is flattened. There is resonance at the apex, diminished at and below the fifth rib anteriorly. Flatness posteriorly with absence of fremitus. He expectorates much purulent sputum dark in color with whitish flakes. No tubercle bacilli are found in it.

Third admission.

The present trouble dates from September 22, 1896. For two weeks previous he had had a slight cough, but on this date at night he coughed up a large quantity of greenish pus which at times poured out of his mouth. Violent cough and dyspnœa developed. September 25 he spat up two spittoonsful of blood-tinged greyish-yellow material.

PHYSICAL EXAMINATION.—The thorax is rather emaciated; there is marked depression under the right clavicle, and expansion is much more marked on the left side. There is no elevation of the ribs of the right side on respiration. Vocal fremitus is slightly increased in the right upper front, is much diminished in the right lower front and back. The percussion note is hyperresonant over the right front except at the apex where the note is higher pitched than normal. On the right front metallic cracked-pot resonance is heard over the upper two ribs. Flatness begins at the fourth rib in front and extends to the costal margin. There is flatness everywhere behind.

Auscultation.—Left side. There are dry râles everywhere, sonorous and sibilant at the apex. Right side. There is an area about the size of a silver dollar at which the breath sounds are amphoric and whispered pectoriloquy is present. Below this the breath sounds are almost absent. In the upper region there are many crackling, sonorous and sibilant râles.

Heart.—The apex beat is under the 5th rib 2 cm. inside the mammary line.

The sputum on admission was of a musty odor, but, September 28, of a horribly offensive odor. No tubercle bacilli were found, but many streptococci.

Transferred to surgical side October 2, 1896.

Discharged November 13, 1896.

Surg. No. 5890.

DIAGNOSIS.—*Recurrent empyema following gangrene of the lung.*

OPERATION.—October 2, 1896. Excision of a rib, incision into an empyema, drainage.

300 cc. of thick creamy pus were removed. The cavity was irrigated each day with 1:1000 $KMnO_4$, followed by half saturated boric acid.

October 15. The patient's condition has gradually improved. Each day about 15-20 cc. of pus drain from the chest. It is not foul smelling. There is no sputum. The lung is expanded by Wolff's bottles.

November 13. Patient discharged much improved.

Urine negative.

Temperature normal since October 28. Before then it was slightly elevated (not above 100°) and irregular.

Dr. Osler, in reporting this case, October 17, 1896, said: "The case is notable because of its origin and duration. It was probably an empyema following pneumonia and then the common sequence of perforation into the lung with the production of a pneumothorax." Operation was performed and the patient was discharged in excellent condition but returned a little over two years later because of much purulent and bloody sputum. Again he left the hospital to return eighteen months later with the history that he had expectorated a great amount of pus, first of musty then of horribly offensive odor, and the diagnosis of recurrent empyema following gangrene of the lung was made. Again operation was performed on the right side and the patient discharged six weeks later much improved.

CASE 41.—*Pyopneumothorax from the rupture of an empyema through the lung; operation; recovery; recurrence of the empyema; rupture; recovery.*

Charles M. D., aged 25, white. **Surg. No. 9783.**

Admitted December 16, 1899.

Discharged January 15, 1900.

DIAGNOSIS.—*Pyopneumothorax.*

The patient COMPLAINS of pleurisy.

FAMILY HISTORY is negative.

PAST HISTORY is unimportant.

PRESENT ILLNESS began November 28 with a sharp pain on the right side so that he could hardly breathe. There was no history of previous exposure or wetting. The patient walked home feeling perfectly well except for this pain. Went to bed immediately and was treated for pleurisy. The cough set in several days after the onset. There was fever a few hours after the pain. Deep respiration has been impossible. After the patient had been in bed a week he began to have severe coughing spells which prevented his sleeping. The sputum during these paroxysms of cough was very foul smelling, of a yellowish-green color and large in amount. All these symptoms excepting the pain gradually grew worse until his admission to the hospital. His physician aspirated him December 15, obtaining about a pint of foul-smelling creamy pus. The patient has lost considerable weight.

PHYSICAL EXAMINATION.—The chest is well formed. The two sides are about equal. On quiet respiration both sides move equally above but the left more than the right below. Percussion: The note is hyperresonant over the left chest in the front and back; also over the upper front and

upper back of the right chest. Is dull over the lower front and back. On auscultation the breath sounds are almost absent over the dull area of the right side. Operation December 16, 1899, Dr. Cushing. The desperate condition of the patient rendered operation imperative. His cyanosis is extreme; the pus is running from the mouth. The 8th rib was excised practically without anæsthesia. On opening the pleura a pneumothorax cavity was found containing a large amount of foul-smelling pus.

December 17. The patient is more comfortable. His cyanosis has been relieved by oxygen inhalations.

December 24. The patient's condition has steadily improved. The dyspnœa is much less; the appetite is good.

December 28. The patient began to use Wolff's bottles to expand the lungs. Below the point of resection of the ribs there is a swelling which has gradually increased in size. In its center is the point at which an aspirating needle has been previously introduced. The swelling is tender and fluctuation is present. The abscess was opened under cocaine and some foul-smelling pus removed, the same as from the empyema cavity.

December 31. The general condition of the patient continues to improve. The abscess cavity is healing slowly. The cavity is draining well.

January 15, 1900. The patient goes out to-day in very good condition. There is still a slight discharge from the pleural cavity but this is almost obliterated. The patient's lung on the affected side apparently comes down as low as the angle of the scapula.

February 13, 1900. The patient has gained 25 pounds since leaving the hospital and now looks in perfect health. The tube still remains in the right side. There is rather marked deformity, especially in the axilla. The expansion in the upper right front is good but below the nipple is tympanitic. In the back there is quite marked scoliosis with convexity to the left. On percussion the left side is found somewhat hyperresonant. Resonance on the right side very good as far as the nipple in front and to the angle of the scapula behind. Below, it is markedly impaired. On auscultation the breath sounds are somewhat exaggerated all over the left side; on the right they are very well heard but have a slightly muffled character suggesting thickened pleura. Below the angle of the scapula they become quite indistinct.

The urine is negative. The temperature on admission was 104.4°. After operation it fell gradually, reaching normal on December 24. It was slightly elevated until the time of his discharge.

Second admission, January 22, 1901. Transferred to the medical side January 24.

Clinical Diagnosis.—*Bronchiectasis.*—Patient thinks he has persistence of the empyema. Eight weeks after the operation for empyema the tubes were removed and the sinus completely healed. The patient remained perfectly well for a year.

Present Illness.—About ten days ago felt as if he had the "grippe," began to cough and spit up phlegm January 19. He has had no illness

since the previous attack. At present there is no pain. There is a little soreness, however, over the operation wound.

PHYSICAL EXAMINATION.—The left lung is clear. On the right side there is apparently thickened pleura. Resonance over this side is somewhat diminished. The breath sounds are faint; vocal fremitus is diminished. The intercostal spaces are equal; expansion is equal. There is no movable dulness. The patient has a very profuse mucopurulent sputum, coughs violently at frequent intervals. The sputum is thick and tenacious.

The urine is negative. Temperature from 99.4° to 101.4°. Transferred to the medical side.

Transferred from the medical side February 2, 1901.

Discharged February 15, 1901.

CLINICAL DIAGNOSIS.—*Empyema necessitatis.*

While on the medical side on the 28th of January there was a rupture of an empyemic abscess and the escape of a large amount of purulent matter. The cough and the expectoration diminished at once and were absent for about 12 hours after the rupture. There has been a slight continuous discharge from the sinus on the right side.

February 13. The discharge has steadily decreased. The sinus is very small. There is good lung resonance to the level of the sinus. Dulness below. There is no cough, no expectoration. Patient discharged.

In this case the acute pleurisy of the right side began suddenly November 28, 1899. Aspiration December 15 showed pus present, hence he came at once to the hospital, where the operation was done at once and while the empyema was discharging through the lung. The operation showed the cavity of the chest to be a pneumothorax. The patient was discharged just one month later and in another month was quite well. Just one year later his old trouble, "following the grippe," he said, returned, and the empyema necessitatis rupturing he recovered. It is then pyopneumothorax from the perforation of an empyema through the lung, but the fact should be remembered that before admission the patient had been aspirated and hence the etiology of the pneumothorax is in doubt. Dr. Cushing's note made at the time of operation was "The pneumothorax apparently occurred shortly after the patient's entrance."

TRAUMATIC PNEUMOTHORAX.

Traumatic pneumothorax is a form seldom mentioned in reports from medical clinics. It is through the kindness of Dr. Halsted that we are able to add the following cases to our report.

Punctured Wounds of the Lung.—The first to recognize this form was the author of the Hippocratic passages who, speaking of emphy-

sema following ruptures and contusions of the flesh, uses the succussion plash for diagnostic purposes. The cases which Celsus mentions certainly belong to the same group. Military surgeons were early acquainted with this form, for Purmann,[4] arguing for an operation for empyema, states that military surgeons had long known that the penetration of air into the pleural cavity caused no inconvenience. The case of Littré [9] is of particular interest since there is no doubt that the condition followed the sword thrust, and in no other case is the emphysema recorded of so extreme a grade. For the possible cases of pneumothorax due to stab wounds received in duels and the benefits of aspiration in their treatment, the remarks of Marquest de la Motte [13] are suitable for speculation. The physics governing the lung in such cases are quite well stated by Hewson.[24] The difficulties in the formation are well stated by Benjamin Bell,[34] who says that they must be small and oblique if pneumothorax is to result. It was John Bell [35] who stated the mechanism of the process much more correctly than did his predecessors and whose review of the physics of the respiration and of the collapse of the lung is even now worth the reading. Butlin's [150] two cases, due to rupture of the lungs and pseudopneumothorax, are of interest. Demarquay [129] states that penetrating wounds of the lungs with difficulty cause pneumothorax and that the lung must be pushed away from the chest wall with some force in order to produce the condition. Biach [171] states that of 21 penetrating wounds of the chest in the Vienna hospitals in none did pneumothorax result.

Pneumothorax due to Fractured Ribs.—One of the first possible cases on record due to fractured ribs is Méry's;[10] another is that of Le Dran,[12] and in this case the diagnosis is not certain. A very rare case is reported in the *Medical Examiner*.[73] in which the fracture of the clavicle was the cause of the pneumothorax. Billroth (Riedinger [220]) considers this a rare cause and states that in but four of 54 cases of non-fatal fracture of the ribs was pneumothorax present. Turner,[221] reviewing the reports of the St. Thomas Hospital of London, states that in 237 cases with fractured ribs there were 28 deaths; 4 cases presented pneumothorax and all four were among the fatal cases; nevertheless in 28 cases was hæmoptysis present and in 25 emphysema, hence in these cases the lung was certainly injured. Turner [221] adds one case with recovery and considers it a very rare case. Among other cases due to fracture of the ribs may be mentioned Saussier's,[84] Hotte,[92] and Janssen,[98] whose case is interesting; a man fell 50 feet; he received no immediate injury. Five days later sudden pneumothorax developed and he died that night. At autopsy four fractured ribs were found. Biach,[171] of 132 cases of fractured clavicle found pneumothorax was present but in two. Hutchinson (West [188]) states that he saw many cases following fractured ribs. Page mentioned one. Auquier [175] mentions the case of a man who was run over and as a result fracture of the ribs. This case is of particular interest since pneumothorax without subcutaneous emphysema was present in one side, subcutaneous emphysema without pneumothorax present on the other side. Thus two possible results of a wound of the lung from fractured rib were present in this

patient. He mentions a number of references to literature not found ordinarily. Lucas [256] reports one case.

OTIS. Pneumothorax. *The Medical and Surgical History of the War of the Rebellion*, Part I, Vol. II.

In the vast number of chest wounds during the war of the Rebellion pneumothorax was a troublesome complication in less than half a dozen cases.

CASE 42.—*Traumatic hæmopneumothorax of the right side; general emphysema; death; autopsy.*

B. B., white, aged 11. **Surg. No. 3837.**

Admitted January 28, 1895.

Died February 6, 1895.

Diagnosis.—*Hæmopneumothorax, traumatic; general emphysema.*

The patient was brought into the accident department with a history that he was knocked down and pushed along the track by a coke cart, the wheel striking him about the tenth and eleventh ribs of the right side but not completely running over his body. He was evidently suffering great pain; was conscious; pulse 116, weak; respirations 36, labored. There was an accumulation of mucus in the throat and râles over the chest. There was emphysema over the right chest, an abraded contused wound over the lower ribs and scratch marks over the left side of the chest near the sternum at about the 7th rib. There was muscle spasm over the entire abdomen, pain on examination, especially over the lower right thorax. The patient was stimulated and sent to the ward. The face, neck and entire thorax became emphysematous, respirations became more laborious. Breath sounds are not marked on the right side; are distinct on the left. The right thorax was aspirated this evening; nothing obtained. There was hyperresonance over the entire chest.

January 29. All signs are masked by the emphysema. There is movement only at the apices of the lungs; movable dulness on percussion. Exaggerated breath sounds over the left, diminished over the right. Percussion note tympanitic. There is general interstitial emphysema over the face, the thorax and the abdomen. The left side of the face is more swollen than the right.

January 29. "Operation, resection of the 6th rib in the posterior axillary line; evacuation of about 400-500 cc. of blood from a hæmopneumothorax. The wound is packed with bismuth gauze into the pleural cavity. The skin wound partly closed with silver wire." The tissues were found infiltrated with blood and air. After resecting 3½ cm. of the 6th rib the pleural cavity was aspirated; no fluid was found. The pleura was incised when air and pure blood gushed out. The opening was quickly closed and again opened, allowing a little more blood to flow out, the patient being on his back. The cavity was packed with gauze and the vessels in the intercostal muscles ligated. The packing was then removed and the chest examined. It was found that most of the blood had been removed; that

the lungs could be seen and felt. Bismuth gauze was then packed into the pleural cavity, completely plugging the opening and the skin partly closed.

January 30. The breathing is easier, the sounds are definite and bronchial over both sides; no cough. Immediately after operation the emphysema began to decrease.

February 2. The general condition very good; the emphysema has much diminished. There is very little blood staining of the dressings. Respiration easy.

February 3. Fresh bismuth gauze introduced. The lung expands well and can be seen in the wound.

Coverslips from the blood discharged at the operation showed no organisms.

February 6. Temperature 101 on admission; rose to 104 and remained elevated. To-day it is 101. The pulse has been slowly rising and is to-day 168. The patient died this morning.

Autopsy No. 623.

Anatomical Diagnosis.—*Accident; rupture of lung and liver. Subcutaneous emphysema. Operation wound. Bronchitis and bronchopneumonia.*

Body 125 cm. long, well nourished; surface sallow. In the subcutaneous tissue on the left side of abdomen is a doughy mass which on incision contains air.

Left lung collapses. There are a few ecchymoses beneath the pleura. The vessels are free. The bronchi contain muco-pus. On section there is no consolidation but on pressure pus escapes from the smaller bronchi.

Right lung.—There is an incision posteriorly which puts the right pleura in communication with the pleural cavity; gauze projects into the cavity. There is no excess of fluid but the pleura is injected. The apex of the lung is bound by fine adhesions to thorax wall. In these adhesions, on section of lung, calcified nodules are found; these are also found in the lymph glands. The lower lobe of the lung is compressed and is covered by fibrinous exudate. Posteriorly the lower lobe is ruptured; the edges of the rupture are ragged and extend half across the lung and to a depth of 5 mm. On section of this part, near root of lung, the tissue is found to be hæmorrhagic. The whole of the lobe is consolidated by the confluence of a number of bronchopneumonic patches. Between the elevated pale granular patches the lung tissue is moist-looking, opaque grey in color and smooth. From the cut bronchi a creamy pus wells up. The pleura over this part of the lung is thickened by old adhesions and covered by a fresh exudate.

Liver.—The under surface of the liver on the right side for a space of 3 x 3 cm. is roughened; the capsule is deficient and there is a superficial loss of substance; this is near the free edge and the right border. Running obliquely across the right lobe of the liver, for a distance of 11 cm., is a linear depression 1 cm. in width, over which at the right border the

capsule is thickened. On section this is found to correspond with a hæmorrhagic line which is 4 cm. in depth. On each side the liver substance is congested. This line is at its furthest point 4 cm. from the upper (superior) surface of the liver. On making transverse sections of this scar, opaque yellowish-white foci, resembling coagulated tissue, are occasionally met with in the red line and on each side of it. The liver presents a rather peculiar dark color, almost chocolate.

The general emphysema was evidence that the lung was ruptured; the hyperresonant note and distant breath sounds that air was in the pleural cavity. This was confirmed at operation, on which occasion air and blood gushed out. The blood was evacuated.

CASE 43.

Charles H., colored, aged 20 years. **Surg. No. 7089.**

Admitted November 2, 1897.

Discharged November 15, 1897.

DIAGNOSIS.—*Stab wounds of the left pleural cavity and arm. Perforation of the pleura.*

At the 3rd interspace is a stab wound about 1 inch long, which admits a probe into the pleural cavity. This and his other wounds are closed with silk sutures. Considerable blood oozed from this wound. There is resonance over this side of the chest but flatness at the base. The breath sounds are jerky and shallow with a to-and-fro friction at the base. Subcutaneous emphysema developed about the wound. There is no bloody expectoration. The pain on deep breathing is severe, and respiration is chiefly abdominal; 48 to the minute.

November 3. Respirations 40, abdominal, painful. No distinct area of dulness is made out. The breath sounds are everywhere harsh, no friction rub is heard.

November 4. The sputum is bloody.

November 5. Temperature, pulse and respiration are all diminished; the patient is comfortable. Just around the wound can be found a slight area of bronchial respiration. The wounds are healing per primam.

November 15. Discharged well.

For the first two days the temperature ranged between 100° and 101.4°, then fell to normal.

CASE 44.

C. W. S., white, 15 years old. **Surg. No. 7587.**

Admitted April 8, 1898.

Discharged April 13, 1898.

DIAGNOSIS.—*Stab wound of chest wall.*

The boy was admitted 15 minutes after the injury with a stab wound of the left chest in the anterior axillary line by a penknife. He chased his assailant, running 50 to 60 feet before he realized his injury

and then felt as if he received an electric shock. Temperature 98.4, pulse 100. There is subcutaneous emphysema throughout the axilla. The air can be heard leaving the wound on breathing. The boy states that he coughed and spat up blood immediately after the injury. There was no pain for about ten minutes; no dyspnœa.

The left side of the chest is decidedly larger than the right and expands much less on respiration, which is accompanied by a dull cutting pain. Over the left front there is diminished fremitus and hyperresonance on percussion. There is no cardiac dulness. Throughout the left back is subcutaneous emphysema. The both backs are about the same on percussion and palpation of the fremitus. Over the left front and back the breath sounds are distant.

The patient was discharged April 13. The subcutaneous emphysema disappeared yesterday. The tympanic quality to the percussion note has disappeared, the two sides expand equally. The temperature has been irregular, from 98° to 100.8°.

February 9, 1899. The patient has had no symptoms since leaving the hospital. Both sides of the chest are equal.

CASE 45.

Joe V., white. **Surg. No. 7873.**

Admitted June 25 1898.

Discharged July 7, 1898.

DIAGNOSIS.—*Stab wound of the right pleura.*

The patient was admitted through the accident department. He gives a history of falling on a stick, causing a lacerated wound of the chest. This happened a few hours ago. On the right side, below and internal to the costal margin, at about the juncture of the cartilages of the 6th and 7th ribs, is a wound 5 cm. long. This wound is deep and passes into the chest cavity. There is marked sucking in of air on respiration. There is no crepitus or broken rib to be made out.

The wound was packed with bismuth gauze and the patient sent to bed. No râles were found on auscultation. The sucking in of air, however, can be heard. Temperature 101.5.

June 26. The patient is comfortable; temperature 99.5; no pain in the wounds.

July 2. The edge of the wound for some distance above has an erysipeloid appearance. The skin incision has been opened. A straw-colored serum exuded. The wound was packed. No complications followed. There was no evidence of blood in the pleural cavity.

The patient was discharged July 7.

CASE 46.

Charles C., white, 26 years old. **Surg. No. 5026.**

Admitted January 12, 1896.

Discharged January 30, 1896.

Diagnosis.—*Pistol shot wound of chest; traumatic pneumothorax; traumatic pleurisy.*

On admission the respirations were 30 to the minute, very shallow. Pulse 106.

Beginning at the 6th rib on the left side and extending anteriorly almost to the sternum, downwards nearly to the costal margin and posteriorly to the posterior axillary line is an area which is hyperresonant on percussion and over which no breath sounds are heard. With the patient on the right side the area of tympany extends to the spine. Above, the percussion note is slightly flattened with diminished breath sounds.

The wound of entrance is 1 cm. in diameter with ragged dark edges and situated 1 cm. posterior to the posterior axillary line between the 8th and 9th ribs. There is subcutaneous emphysema limited by the spine, the angle of the scapula, the posterior axillary line and the costal margin. With the patient on his back there is flatness below the posterior axillary line; lying on his right side this area is tympanitic.

The patient has some cough with bloody sputum.

There is considerable rigidity of the abdominal muscles.

January 14. There is no distension of the abdomen, no tenderness on palpation, the muscle spasm is less. There is a tympanitic area extending from the 7th rib to the costal margin and from the sternum to the axillary line. From the posterior axillary line between the 7th and 10th ribs and extending to within one inch of the spine there is now complete dulness. Breath sounds are not heard over this area.

January 19. The condition has improved. The area of dulness has diminished, the breath sounds are more distinct, no adventitious sounds. Breathing to-day causes no pain.

January 22. The temperature to-day rose to 102.° There was no chill or sweating. The dulness is much more distinct and extends nearly around to the nipple. There are a few moist râles on inspiration. No pain on respiration.

January 25. Temperature is normal, no cough or expectoration to-day. The chest is nearly clear.

January 30. The chest is entirely clear. The patient is discharged.

The temperature on admission was 99.4°. It rose, reaching 102° January 14. It fell gradually and rose again on January 22.

CASE 47.

Owen W. Autopsy No. 1383.

Anatomical Diagnosis.—*Fracture of the scapula and ribs on right side (2nd to 7th, inclusive). Perforation of pleura and lung. Pleurisy. Bronchopneumonia. Bronchitis. Cavity in lung, right. Cloudy degeneration of kidneys. Congenital defect in interventricular septum of heart. Fibrous myocarditis.*

Length of body 156 cm. Body of a well-nourished, well-for- knife. He
Rigor-mortis well marked. Liver-mortis of dependent parts zed his med man.
The right

eye-ball is apparently absent. Abdomen is slightly distended and there is blue discoloration on either side. Blue discoloration, having appearance of bruise, on right thumb and adjacent metacarpal bone. There is reddish discoloration of skin, irregularly distributed over sternum and left side of chest.

In the 8th and 9th interspaces, at their most anterior extremity, the muscle is infiltrated slightly with blood. Tissue immediately behind sternum shows a few small areas of blood infiltration. The left lung is bound to the chest by very numerous fibrous adhesions. Right lung similarly bound.

Left lung is voluminous. On section lung is of a dark red color, pigmentation abundant. The surface is very moist. In the lower part of the upper lobe, immediately above lower surface, are scattered areas, felt better than seen, which have a brighter red color than the surrounding lung substance and a firm, granular appearance. Scattered throughout lower lobe, least abundant in upper part, are similar areas of consolidation, having a diameter of about 5 mm. Bronchi are deeply injected and contain creamy white fluid of a purulent appearance.

Ribs.—The second right rib is broken about 3 cm. in front of its angle. The anterior end has passed behind the posterior, whose jagged end has perforated the pleura. The 3rd and 4th ribs are broken at about the same point. A fragment of 4th rib projects directly inward. The 5th rib is broken about at its angle. The 6th and 7th ribs are broken about 6 mm. from vertebral column. 8th and 9th are intact. The intercostal tissue, especially in the neighborhood of third rib, is infiltrated with blood. In parietal pleura, corresponding to 3rd and 4th ribs, are perforations extending into lung substance, the pleura being here adherent.

Right lung.—Greater part of upper lobe is crepitant. The outer part in neighborhood of perforations, before mentioned, is firm in consistence and contains very little air, portion cut out sinking in water. On section it is of a homogeneous deep red color. This laxly consolidated portion involves about one-half the lobe. The middle lobe is crepitant. Greater part of lower lobe resembles the consolidated portion of upper, sinking in water. In the lower part are numerous areas, red in color, and with granular surface, resembling those in left lung. Exuding from smaller bronchi is a purulent fluid. Larger bronchi are injected and contain a similar material. Corresponding with the opening in the pleura is an elongated cavity, extending into lung substance 4 x 1.5 cm. The wall has a grey necrotic appearance.

With the kind permission of Dr. Halsted I am permitted to add to the list the following series of surgical cases in which although the lung was quite certainly injured, pneumothorax did not result. The notes in these cases are very much abbreviated.

BULLET WOUNDS.

CASE 1.

Henry L., white, aged 16. **Surg. No. 4762.**

Admitted October 26, 1895.

Discharged November 17, 1895.

DIAGNOSIS.—*Pistol shot wound of the right chest penetrating the lung; traumatic pneumonia.*

On admission the respirations are shallow and limited as much as possible on the wounded side. There is seen a pistol ball wound apparently made by a number 32 bullet. The edges are somewhat darkened in color and blood-stained, are smooth and inverted. The wound is entirely round, some hæmorrhage has taken place. No entrance of air can be heard on inspiration. The respirations are shallow on account of pain. On percussion dulness extends well to the nipple in front and is distinctly higher above the back. On auscultation the respiratory sounds are nearly absent in the lower and posterior portion of the back. Over the mammillary region are moist bubbling râles; vocal fremitus absent in the lower front and back.

October 27. Pulse 120-130; respirations 45; very painful. The dulness over the lower lobe has increased. On percussion it extends to the nipple line in front and to the middle of the scapula behind. On auscultation no breath sounds can be heard in the lower third of the right chest. Above this area and extending to the upper mammillary line mucous bubbling râles can be heard.

October 28. Aspiration in the 7th interspace was attempted but negative.

October 29. The sputum has become less and less blood-stained until this morning it is normal.

November 1. The dulness over the lower portion of the right lung is very much less. Respiratory sounds can be heard over nearly the whole area. The cough is less. Sputum scanty, not blood-stained. Patient comfortable.

November 3. The lung is nearly clear. The cough is very much better. There is no fever.

November 10. The lung is clear, no cough, no discomfort.

November 17. Discharged.

CASE 2.

Mary H., aged 13. **Surg. No. 5453.**

Admitted May 18, 1896.

Discharged May 31, 1896.

DIAGNOSIS.—*Gun-shot wound of the chest perforating the lung; right hæmothorax. The bullet embedded in the cartilage of the 7th rib.*

Remarks.—The bullet entered at the inferior angle of the scapula, passed through the lung and stopped subcutaneously opposite the 7th costal cartilage in front. Hæmothorax developed, gradually disappeared and when examined just one week after being discharged the thorax and lung were absolutely clear.

Past History.—A man shooting at a target with a Flobert rifle was about 40 feet distant. The patient did not fall or lose consciousness at any time after the accident, did not vomit, did not spit up any blood; had no difficulty in breathing at first. The pain from the beginning was referred to the epigastric region; no pain in the abdomen.

Physical Examination four and a half hours later. The wound of entrance is 5 cm. posterior to the posterior axillary line on a level with the angle of the scapula. The tissues for a distance of 10 cm. are distinctly emphysematous. No redness of the skin and no œdema; very little tenderness. The emphysema does not extend over the scapula. It extends as far as the spine of the scapula and downward to the 10th rib forward to the mid-axillary line; respirations 44; pulse 116; temperature 99.8. There is dulness on the right side of the chest beginning at the nipple line and extending around to the spine. The percussion note over the lung above the nipple is good; breath sounds clear; no râles. Over the dull area the breath sounds are absent. She complains of much pain at the umbilicus and upper abdomen. The entire abdominal muscles are rigid but this can be explained by the dyspnœa. Three hours later pulse 108; respirations 46; very shallow. There is abdominal tenderness just below the ensiform. The lower abdomen is not unusually swollen. From the nipple line backwards the dulness over the lung is continuous to the costal margin. Between the nipple and the sternum there is a very little dulness but the note is much flatter over the liver region and above the costal margin, while just below the costal margin in the epigastric region it is tympanitic. Percussion around the nipple and sternum is slightly painful. The bullet can be felt at the juncture of the costal cartilages of the 6th and 7th ribs under the skin.

May 19. All abdominal symptoms have disappeared.

May 21. The supra-mammillary and mammillary regions of both sides of the thorax extend equally, but the region from the 6th rib to the costal margin on the left side extends more than the right. Above the 6th rib on the right the note on percussion is pronounced, tympanitic. This tympany ceases abruptly at the 6th rib and becomes almost flat. This note remains constant for the width of two ribs downward, when it becomes Skodaic. There is movable dulness. Over the liver region is wooden tympany. Vocal fremitus is decreased over the right base. Percussion note becomes flat at the 8th to the 9th rib. The point of maximum impulse of the heart is 11 cm. outside the mammillary line. The temperature was slightly elevated during her stay in the hospital, ranging from 99 to 100. She was discharged well.

CASE 3.

Andrew S., 21, white. **Surg. No. 5692.**

Admitted August 1, 1896.

Discharged September 7, 1896.

Diagnosis.—*Pistol shot wound of thorax. Traumatic (double) pneumonia.*

The patient, with suicidal intent, shot himself with a 32-caliber pistol over the heart region. He came to the hospital six hours later. He is a young man in good physical condition. Just 1 cm. upwards and inwards from the left nipple is a clean cut round bullet hole about 1 cm. in diameter. There was no orifice of exit but the bullet seemed to take an upward and inward course.

Pulse 96. Respiration 38, short and jerky. There is no pericardial murmur. The patient has a bloody expectoration. There is dulness in the lower left chest, very movable over the area immediately about the wound are heard fine moist râles.

There is considerable abdominal muscle spasm.

August 5. The entire left lung and the upper lobe on the right side are consolidated by pneumonia.

August 25. The lungs are entirely clear; the breath sounds normal and vesicular.

The temperature from admission till August 4 was between 100 and 102°. On August 5 it rose and till August 15 was remarkably remittent, ranging from 100-105.3°. It then, with irregular elevations at times, fell, reaching normal August 27.

In February 1899, he reported himself in good health, having felt no results of his wound.

CASE 4.

Ella H., 14, white. **Surg. No. 6595.**

Admitted June 2, 1897.

Discharged June 16, 1897.

DIAGNOSIS.—*Bullet wound in left axilla perforating chest.*

The patient while asleep was shot at 5.30 a. m. on the day of admission. The pain was severe at first, she being hardly able to breathe or speak, but later diminished and she was able to walk to the hospital. Also the dyspnœa was at first severe. The wound bled but little.

PHYSICAL EXAMINATION at 8 a. m. The girl is in good general condition (nutrition, etc.). There is marked dyspnœa; pulse 120.

In the right axilla is a blackened wound of entrance, 1 by 1½ cm. in size, below the margin of the pectoralis major at the level of the 3rd rib, surrounded by a powder burn. There is no emphysema or any adventitious sounds on auscultation. On the back, just below the angle of the scapula, the bullet could be felt beneath the skin. It was later removed. No area of dulness could be determined.

3 p. m. The dyspnœa has disappeared. The patient is very comfortable. There is some pain on moving and on deep inspiration.

June 3. The patient nad a comfortable night. Temperature 99.2°; pulse 116; respirations 40. There is no emphysema nor physical signs of lung perforation.

CASE 5.

Gus. B., white, 32 years. **Surg. No. 6862.**

Admitted August 25, 1897.

Discharged August 28, 1897.

DIAGNOSIS.—*Gun-shot wound of chest (penetrating). Hæmoptysis. Emphysema.*

The patient was admitted at 12 m. about fourteen hours after the injury. He was shot from a large revolver in the chest. Immediately blood gushed from his mouth and continued to flow for at least one-half an hour. It then stopped but soon began again. This morning the bloody expectoration ceased.

PHYSICAL EXAMINATION.—The patient is lying comfortably in bed. Pulse 92 to the minute, respiration 40, temperature 101°.

In the 1st left interspace 2 inches from the sternal margin is the bullet wound. There is some swelling about it, which is quite tender. There is spasm of the pectoral muscles. Both sides of the chest expand evenly with respiration. The left lung is clear in front; behind, just above the angle of the clavicle, there is some swelling of the superficial tissues. Here it is very tender to the touch and on auscultation many liquid râles and a crepitus resembling a pleural friction rub, may be heard. The lung is elsewhere clear. The right lung is clear. The heart is negative. The emphysema extended over the scapula. The bullet may be felt just over the blade of the scapula. This was removed under cocaine. The sputum is blood-stained.

August 28.—There is no longer cough or expectoration. The emphysema has disappeared. There are no subjective symptoms.

The temperature for the first two days ranged from 100° to 101.3°. It then fell to normal.

CASE 6.

Powell M., white, aged 28. **Surg. No. 7,054.**

Admitted April 28, 1898.

Discharged September 20, 1898.

DIAGNOSIS.—*Bullet wound of the chest; hæmothorax.*

The patient was admitted with a history of having shot himself in the left chest a few hours before admission. 15 cm. to the outer side and slightly below the nipple is the wound of entrance with a powder burn about it. It looks as large as a five-cent piece. There is a superficial burn of the skin about this for a radius of 4 or 5 cm. The patient complains of pain, especially on inspiration, at the left costal margin in the mammillary line and some shortness of breath. The pulse is 120. Under cocaine an incision was made along the track of the wound and the bullet removed. The patient has neither vomited nor coughed up any blood. There is considerable abdominal rigidity; no movable dulness; no tenderness. He complains of slight nausea. He was prepared for immediate laparotomy. An exploratory operation was performed this afternoon, excision in a portion of the 6th rib. On opening the abdomen no wound could be found. The wound in the pleura was packed.

April 28. There is a slight friction rub over the left præsternal region, thought to be pleuropericardial.

April 30. The patient is somewhat cyanosed; pulse 105; respirations 27. The left chest moves somewhat less on inspiration. There is considerable emphysema about the wound. The emphysema extends almost to the median line in the back. The left chest behind is dull on percussion. The voice sounds are absent. There is no evidence of intra-consolidation. The patient is coughing considerably and raises masses of bloody phlegm.

May 1. The emphysema is still present. The whole left back is somewhat dull with slightly diminished tactile fremitus. The voice sounds are heard faintly over the whole side. There are no signs of consolidation. Along the costal margin of the posterior axilla is new leathery friction rub. The patient is still somewhat cyanosed; general condition is excellent. He coughs up rusty tenacious mucus, containing blood.

May 7. The temperature has varied from 101° to 102°. He complains of much pain in the left side, especially on coughing. The cough has improved. There is no more blood in the sputum. There are distinct signs of consolidation reaching to the angle of the scapula behind and forward to the posterior axillary line. There is whispering bronchophony, tubular respiration, dulness, etc.; there is much diminution of respiratory movement. The patient breathes with a respiratory grunt at the beginning of expiration. The alæ nasi move.

May 8. There is loud tympany in the upper left front, becoming dull as one passes into the axilla and completely flat below the spine of the scapula. Vocal fremitus in this area is absent. In front the respiratory sounds are extremely feeble except at the extreme apex. In the flat area in the back the respiration is almost absent and has a distinctly tympanitic quality. The voice sounds have a nasal quality and distinct bronchophony. A needle was inserted in the 8th space of the left axillary line and 100 cc. of a bloody fluid obtained.

May 26. The patient has improved much. The chest is contracting somewhat.

May 30. The condition of the chest is the same. The chest has been irrigated several times.

June 15. The bullet is palpable in the 10th space just inside the scapular line.

July 16. The patient has been slowly improving. Last night, however, for some unknown reason, his temperature rose to 102.6°. The cavity is daily irrigated with boric acid.

August 3, 1898. Operation. Two ribs were resected, enlarging the opening into the pleural cavity.

September 4. The whole left chest is very much flattened and depressed; there is very little movement on inspiration. The wound corresponding to the point of entrance which was explored on admission was closed. That subsequently made for drainage has closed down to the sinus and is represented by a deep depression where two ribs have sunken. The respiratory sounds at present over the left chest are well heard except in the region of the point of drainage. The sinus runs 15 cm. upwards.

The chief cavity seems to be shortly within the opening and above it corresponding to the axillary region. This cavity holds 100 cc. During admission the temperature was quite regular, varying from 101° to 102° until May 5. Then from 101° to 103° until May 11 when it rose to 104.8°. It remained elevated until June 13, when it reached normal.

On July 15 it rose again but on the 17th was again normal and remained so.

CASE 7.

Michael M., white, aged 16. **Surg. No. 7855.**

Admitted July 3, 1898.

Discharged September 10, 1898.

DIAGNOSIS.—*Gun shot wound of the chest (left side). No effusion; slight dry pleurisy.*

The patient was accidentally shot from a pistol ten feet distant. He was not short of breath nor did he cough up any blood. He had some pain in the left shoulder and arm, also in the abdomen. He was brought to the hospital two hours later, where the wound was cleansed and dressed. While being dressed in the dispensary slight bubbling was noticed through the blood issuing through the hole in the chest.

PHYSICAL EXAMINATION 24 hours later. Between the second and third ribs close to the left border of the sternum is the wound of entrance. There is no reddening about the edge of the wound. Under the left arm in the anterior axillary line there is considerable thickening of the 4th rib and some œdema; crackling is felt on palpation of this region. This area is not movable, is not tender on pressure, suggesting the probability of the bullet being embedded at this point of the rib. The right chest moves more than the left. There is no especial fulness on the left side. Respirations are 30. Auscultation shows a number of pleural crackling râles at the bases in front and behind in the axilla, extending well up to the 2nd rib. There is some relative dulness in this region. The breath sounds are harsh but well heard. As yet there is no sign of fluid. The heart sounds are clear.

July 8. There is little or no pain although the temperature without apparent cause reached 101°. Chest examination is negative

July 10. The temperature is normal. The patient was discharged at his own request.

CASE 8.

Wilbur H., white, aged 14. **Surgical Number 8073.**

Admitted September 5, 1898.

Discharged September 19, 1898.

DIAGNOSIS.—*Bullet wound of the back perforating the lung.*

On admission the patient states that about four hours ago he was accidentally shot in the back with a 22-caliber pistol which was only about four feet distant. Examination shows a small wound of entrance over the right scapula. There is no pain on moving the arm or placing it above the

head. The bullet cannot be palpated. The wound was thoroughly cleansed and dressed.

September 8. There have been no symptoms since entrance. This morning he had a sudden pain along the right costal margin. Respiration 44; considerable dyspnœa.

Examination of the chest negative.

September 10. Percussion is flat at the angle of the scapula; behind at the 4th rib in front no movable dulness. Leucocytes, 12,400.

September 12. There is very little movement on the right side of the chest. Dulness remains at the angle of the scapula. Over the dull area, especially near the spine, is prolonged expiration and some fine crackling râles following cough. The voice sounds are well heard. Vocal fremitus is absent over the dull area.

September 19. The dulness has quite disappeared. There is slight feebleness of the breath sounds over the right lower back.

Discharged.

February 10, 1899. The chest is clear. There is no evidence of the old lesion. The urine is negative. The temperature was slightly elevated during his stay in the hospital. The highest point was on admission, 100.2°.

CASE 9.

Lawrence H., white, 10 yrs. old. **Surg. No. 11,191.**

Admitted Nov. 13, 1900.

Discharged Nov. 29, 1900.

Diagnosis.—*Bullet wound of chest.*

The bullet entered under the left clavicle and lodged under the skin near the spine of the scapula. There were no symptoms except some transitory pain. The bullet was recovered by incision.

The accident happened six hours before admission. He was able to walk home in two hours and has been driven 9½ miles to the hospital. He has not expectorated or vomited blood.

Physical Examination.—The boy is in good condition, pulse 128, temperature 100.5. The wound of entrance is just below the left clavicle, 2 cm. from the mid-line. There is no burning of the skin, no bleeding from the wound, no emphysema. The chest is resonant and the breath sounds heard throughout. The apex beat is in the 4th i. s. inside the nipple line. Above the spine of the left scapula is an area of subcutaneous emphysema 10 cm. in diameter, over which the skin is bluish. The emphysema extends for some little distance down over the body of the scapula. No foreign body, no fractured bone can be felt. The percussion note and breath sounds over the left back are clear.

Nov. 18. The pain has disappeared, the swelling gradually subsided. There has been no spitting of blood, no lung signs.

Nov. 24. An incision 2 cm. long was made in the suprascapular region. The bullet and several pieces of clothing were removed.

Nov. 28. The wound has healed per primam.

Nov. 29. Discharged.

The temperature on the first day, 100.5° to 101°, remained irregularly elevated during his stay but gradually fell towards normal.

STAB WOUNDS.

CASE 10.

Charles Q., colored, aged 28. **Surg. No. 4843.**

Admitted Nov. 15, 1895.

Discharged Dec. 16, 1895.

CLINICAL DIAGNOSIS.—*Stab wound perforating the left pleural cavity.*

The stab wound was inflicted yesterday. The wound bled profusely, saturating his clothes. On admission the wound was still bleeding. It was about 2 cm. long in the 7th interspace of the lower front chest. The result of this injury was not pneumothorax. The pericardium seems to have been perforated for the heart sounds are accompanied by a friction rub loud and snapping and close to the ear. An occasional splashing râle is heard, so loud that the normal heart sounds are almost entirely obscured, and is transmitted for a short distance in all directions about the heart. Pulse 92 to the minute, rather full; respirations 30 and shallow. The patient complains of some pain across the chest over the heart.

Abdomen negative. Temperature 101.2°.

November 16. Temperature 101.1°; pulse 96. There is the same rasping murmur coincident with systole and diastole of the heart and present when the patient is not breathing.

November 28. The wound was dressed. After removing the packing a small cavity was found completely walled in. The posterior walls of the cavity seem to be formed of the pericardium and there is extensive pulsation synchronous with the heart. Patient discharged.

CASE 11.

Emanuel Y., white, 28 yrs. old. **Surg. No. 6383.**

Admitted March 20, 1897.

Discharged March 27, 1897.

DIAGNOSIS.—*Multiple incised wounds. One punctured wound.*

The patient was admitted one hour after he received several stab wounds with a pocket knife, on the shoulder, face, hand and axilla.

The only wound of consequence was on the left side mid-axillary line, 7 cm. below and outside the nipple. This directed downwards and inwards, chipping off a small piece of rib, enters the pleural cavity, perhaps also the peritoneal.

This side of the chest moves less than the right; below there is dulness on percussion and the breath sounds are distant. The wound was lightly closed. The patient recovered rapidly. The temperature was at first raised 99.5° to 100.8°, but gradually fell to normal. The wound healed per primam, and on the eighth day he was discharged, well.

CASE 12.

William G., white, 25 years. **Surg. No. 6504.**

Admitted May 2, 1897.

Discharged Nov. 11, 1897.

DIAGNOSIS.—*Stab wound of the right side penetrating the pleura. Bronchitis.*

Last night at 2 a. m. the patient was stabbed from behind with a pocket knife. The wound is 1¼ cm. long over the 9th rib on the right side, about 4 inches behind the mid-axillary line. There is much pain in the shoulder, some on deep respiration. The tissue about the wound is swollen, there is no emphysema there. No pleuritic friction heard, the breath sounds in that region are normal.

May 3. There is sharp pain in the region of the wound. Respiration is shallow and rapid. The abdominal walls are held rigidly. The wound, which had been closed before admission, is now opened, and in a few minutes dark fluid blood accumulated. No fracture of the ribs are found. The patient has expectorated no blood. The temperature on admission was 97°. It rose in a few hours to 101.3°, and at 4 a. m. to-day was 102.2°. Respiration has been between 28 and 30, the pulse between 100 to 112. There is a little cyanosis. Respiratory sounds are heard about the wound, but none between the wound and the 9th rib. There is no change in the percussion note.

May 5. The swelling about the wound is hard and painful. The right lung is normal. The pleural cavity was explored with a needle but no fluid was found. The percussion note is a little duller than normal over the apex; above the 5th and 6th ribs the respiratory murmur is harsh with slight tubular modification. Below the 6th rib the breath sounds are scarcely heard. All over the left side dry and moist râles are heard. There is no pleuritic crepitus. The cough is somewhat troublesome. The sputum is dark, tenacious, slightly blood-flecked. The apex beat is somewhat outside the nipple line; the sounds are clear.

The left side was aspirated below the 6th rib, but with negative results.

May 9. The temperature is 100.4°, the patient has been comfortable for the past 3 days. The bronchitis has cleared up.

May 11. The patient is discharged. The hæmatoma beneath the skin at the site of the wound has suppurated.

Feb. 1, 1899. The patient is well, has no cough, the scar is soft.

CASE 13.

Joseph P., white, aged 19. **Surg. No. 10,893.**

Admitted September 9, 1900.

Discharged September 10, 1900.

DIAGNOSIS.—*Stab wound of the chest.*

The patient was admitted complaining of pain in the left side of the chest, following a stab wound. This was inflicted with a pen-knife two days ago in the forenoon. The wound was dressed in a neighboring hospital. That

night the patient began to have sharp pain in the left side, especially on coughing or taking a deep breath. He says the pain was inside the chest and not around the wound. He could not sleep that night. This pain continued until yesterday, but this morning feels somewhat better. He had a chill yesterday morning. He has had very little cough and no expectoration.

PHYSICAL EXAMINATION.—A healthy looking man; not anæmic. On the left side of the chest at the top of the 4th rib 6 cm. from the mid-sternal line to the lower border of the 6th rib is a superficial wound, and another one extends along the 6th rib. There is a slight tenderness in the lower part of the first wound. Both are perfectly healthy. Expansion seems to be more marked on the right side. There is no impaired resonance. Vocal fremitus is less easily felt on the left side in the front and axilla. The breath sounds are clear on the right side. On the left side in the lower front and axilla the breath sounds are somewhat distant. In the left axilla there is a slight, friction rub. The right back is clear. The left back low down gives slightly distant breath sounds and a few large moist râles on expiration. The cardiac dulness is normal. Coughing gives considerable pain. The temperature was 100°.

CASE 14.

Marian W., white, 34 yrs. old. **Surg. No. 7260.**

Admitted Dec. 30, 1897.

Discharged Jan. 8, 1898.

DIAGNOSIS.—*Contusion of the left side of the chest. Slight local pleurisy.*

COMPLAINT.—Soreness of left side, increased on taking a deep breath; spitting of blood.

On the night of Dec. 25 she tripped, fell backwards and to the left, striking against the door. She became unconscious. The next morning she was in severe pain, and expectorated a great deal of blood. This has continued till admission, decreasing in intensity. The pain is sharp, in the left lower chest, running from behind forward and accompanied by stiffness of the side. She must take rapid shallow breaths. This has improved much.

Jan. 3. The patient complains only of a sharp sticking pain in the left side, constant, but increased by a deep breath or coughing. The cough has improved, the sputum diminished, it is mucus with an occasional blood speck. The temperature has not been above normal.

PHYSICAL EXAMINATION.—Respiratory movements and percussion notes are normal. Auscultation is normal except at the left base in front and behind are a few fine crackles, and behind at the angle of the scapula are occasional moist râles.

Heart.—Maximal impulse in the 6th space 8 cm. from the mid-line. At the apex a blowing systolic and at the base a soft diastolic murmur are heard.

PNEUMOTHORAX DUE TO CAUSE EXTERNAL TO THE CHEST.

Abscess of the Liver.—The first case reported is that of Lemaire,[n] well reported and evidently free from criticism.

CASE 48.—*Pyopneumothorax from the rupture of an amœbic liver abscess into the lung and pleural cavity; operation; death; autopsy.*

Frank E., white, 42 yrs. old. **Med. No. 5702.**

Admitted Jan. 7, 1896.

Died Feb. 10, 1896.

CLINICAL DIAGNOSIS.—*Amœbic Dysentery. Amœbic Abscess of the liver and lung. Thrombosis of the vena cava inferior. Pneumopyothorax.*

COMPLAINT.—Severe cough.

FAMILY HISTORY good.

PAST HISTORY negative.

PRESENT ILLNESS.—He dates the onset to last September, when chills and fever began, on some days two or three attacks a day, other days having none. These attacks lasted from one-half to two hours. They consisted of severe shaking chills and profuse perspiration. There was no periodicity noted. He has worked but nine days since the onset; has been in bed since Dec. 21. For three days previously he had a diarrhœa.

On Jan. 2 he began to cough, the cough persisting and increasing in severity. On Jan. 3 he first noticed blood in the expectoration and since that time it has been quite profuse and free. He has been unable to sleep owing to the intense coughing. No headache, no nausea, no vomiting, appetite good.

In November he had diarrhœa for a few days.

PHYSICAL EXAMINATION.—Jan. 8 (Dr. Osler). Temperature 102.5°, pulse 108. Heart apex beat in the 5th i. s. just outside the nipple line. Respiration is quiet. Chest well formed, intercostal spaces visible on both sides. No swelling in the right hypochondrium. No area of visible pulsation. Expansion almost equal on both sides, perhaps a little more on the left than right side. Percussion is clear on the right side to the upper margin of the 6th rib, in the axilla to the lower margin of the 5th. On firm percussion there is less resonance from the lower margin of the 4th rib down. Behind the note increases in flatness from the mid-scapular region down. Tactile fremitus well felt, not increased. Breath sounds are feeble at the right base on quiet breathing. There are a few distant crackling râles becoming large on deep breathing and coughing. Breathing is nowhere tubular.

In the lower right axilla the breath sounds are feeble, expiration is a little tubular. On coughing and deep inspiration are heard a few medium sized râles, but they are not nearly so abundant as in the back. Breath sounds are low but clear.

Left side is clear.

Heart negative.

Abdomen.—The liver does not seem enlarged and not sensitive even on deep pressure.

Sputum brownish-red, rusty, looking somewhat like anchovy sauce, yet not quite the brick-red color of this. It consists chiefly of pus cells and red blood corpuscles, a few pigmented alveolar epithelium cells, well marked bilirubin crystals, several fragments of elastic fiber, and amœbæ coli. The rectal tube was passed but no amœba found.

The sputum was examined daily for the next twenty days. The amount varied from 80 to 300 cc. in 24 hours. The character remained the same, anchovy sauce sputum, containing blood, pus, elastic tissue and amœbæ coli. The temperature has been irregularly elevated, several times reaching 104°. There has been no jaundice.

Jan. 27 (Dr. Osler). The patient has not been so well. There is no one point where the abscess can be said to be superficial. There is dulness at the angle of the scapula, the râles are loudest in the lower part of the interscapular region near the spine.

Jan. 31. The patient has grown weaker. The sputum is more watery. He has been wheeled onto the bridge each day.

Feb. 2 (Dr. Thayer). There has been no blood in the sputum for several days. Yesterday, at 2 p. m., the patient had a sudden pain in the right chest, the feeling of oppression and dyspnœa. The temperature last night was 103.2°. This morning the patient is lying on his back coughing constantly; pulse 144. The right side of the chest is markedly full, the intercostal spaces almost obliterated. There is marked throbbing over the cardiac area. The right side of the chest is motionless, vocal fremitus increased. On percussion there is wooden flatness everywhere. On auscultation of the upper front of the chest there is a feeble metallic, somewhat amphoric respiration. A few adventitious sounds. The coin sound is moderately well conveyed. There is complete flatness throughout the back and distant amphoric respiration. Movable dulness and succussion were not tried, since he was so weak.

Transferred to the Surgical side Feb. 2, 1896.

Operation Feb. 2, 1896.

Died Feb. 10, 1896.

The right pleural cavity was opened, about 3 cm. of rib being resected and a large quantity of light brown pus discharged. The discharge remained profuse till the death, and the cough severe.

Feb. 4. The amœbæ coli were found in the stools.

Feb. 10. Yesterday the patient began to have a diarrhœa, passing eleven stools during the day. The stools are watery, containing blood, mucus and numerous amœbæ. The patient died to-day. The temperature has ranged from 100° to 104° and has been very irregular. The urine was negative.

Autopsy No. 769.

Anatomical Diagnosis.—*Amœbic dysentery; amœbic abscess of liver perforating into lung. Fibrino-purulent pleurisy. Pyopneumothorax.*

Perforation of inferior vena cava.. Metastatic lung abscesses due to the S. pyogenes aureus. Metastatic liver abscesses. Thrombosis of inferior cava and right auricle Thrombi in hepatic veins. Congestion of intestines.

Body of a sparely built, much emaciated man. There is no subcutaneous œdema. Rigor-mortis in both extremities. On the right side, over the thorax, an opening 1 x 3 cm. is present which leads into the thoracic cavity of that side. The edges are gaping and covered with a mottled red and grey exudate.

Left lung is insufflated; free from adhesions. Both the upper and lower lobes contain many areas of lobular consolidation, some of which appear just beneath the pleura, which is congested over them, covered with fibrin, and contains small hæmorrhages. Other ones in the depths. On section these areas of consolidation correspond with abscesses which contain a dirty glutinous pus. They vary in size from a hazel nut to an almond, and are found in both lobes. In number they reach ten or twelve in both lobes. The intervening lung substance is congested and insufflated. The bronchi contain muco-pus and are congested. The vessels at the base of the lung are free.

Right side.—Occupying the right lobe of the liver in part is an abscess cavity. It is in the upper part of the lobe, next to the diaphragm and communicates with an abscess cavity in the right lung. The diaphragm is adherent over the liver cavity, so that the lung and liver are in direct communication in this part. On each side of the cavity the liver is adherent to the diaphragm below and the lung adherent to it above. The contents of this cavity is thick, greyish, necrotic looking tissue, greatly disintegrated and indeed would be called thick pus. Some clotted blood is also contained intermingled with this in its more central parts, and softer material of sand-like appearance is also present. The walls of the abscess are dense, evidently fibrous, and around the cavity they are covered with tolerably firm and drier pus-like material. The abscess measures in its entirety 19 x 14 x 9 cm; it extends behind to the capsule of the liver and in front is separated by a narrow zone of liver substance from the capsule and the peritoneal cavity. The wall behind extends to the liver and the inferior cava; a rupture has occurred into the vena cava just about the point of the entrance of the hepatic veins. This rupture is 1.5 mm. in size, and about it the intima of the vessel is lifted up and by the least exertion a grumous material passes into the veins. (Whether or not the ruptured place was made larger by the removal of the liver cannot be said, but it is not improbable). Beginning at a point above the rupture the inferior vena cava contains a decolorized, firm, adherent thrombus, which extends upward and projects into the right auricle, which it about one-third fills. Some of the hepatic veins are thrombosed in parts of their extent, the thrombi in the upper part being decolorized, but deeper in the liver substance they are red and soft.

The lung which surrounds the abscess cavity is firm, grey in color,

œdematous, forming a capsule 2 to 4 cm. in width about the cavity. The lung tissue above this is compressed. The abscess is limited to the lower lobe. The middle and upper lobes are firmer in consistence and more or less compressed. The pleura covering the lung, as well as that on the chest wall, is much thickened and covered with an opaque yellow false membrane, containing fibrin and pus. There is no fluid in this pleural cavity. Below the lung is stuck by recent glutinous but fairly dry adhesions to the chest wall. No perforation can be found in the pleura. The abscess in the lung communicates with bronchi which contain similar contents. Vessels are free.

The lower edge of the liver is a finger's breadth below the costal margin. There are firm adhesions to the diaphragm over the right lobe.

This case is included in the pneumothorax series although the surgical note made at operation says nothing of the presence of air in the pleural cavity. The note is very brief and the omission does not necessarily speak against its presence. At the autopsy no perforation was found.

In favor of pneumothorax the following may be said. The sudden onset of dyspnœa February 2 did not mean the perforation of the liver abscess through the lung, for that occurred one month before. It possibly meant the perforation of the abscess into the pleura, since pus was present at the operation and the signs of February 2 were very different from those of January 27; there was no change January 31. But was air present in the pleura with the pus? We think so. The sudden pain and dyspnœa, the marked fulness of the right side and the moderately well marked coin sound would indicate it.

Hydatid of the Liver.—This cause is well illustrated by Mercier's case.[74] Saussier [78] also reports a very good one.

Gastric Ulcer.—Sturges [145] reports one doubtful case as an illustration. Biach could find but two in the literature. Stephanides [176] adds a possible case.

Cancer of the Stomach.—This was mentioned as a possibility by Saussier. Burrows and Kirkes [105] believe in post-mortem pneumothorax from autodigestion of the stomach.

Ascaris lumbricoidis in the Pleura.—Biach found two such cases in literature. Muller [149a] reported a case of gastric perforation and with three round worms in the pleural cavity.

Perforation of the Intestine.—Cossy [162] reports very rare cases, the result of appendicitis. In each case, however, the pneumothorax was false, the parietal pleura being dissected away from the chest wall and enclosing the gas.

Rupture of the Œsophagus.—The first and for a long time the last case on record was that reported by Boerhaave [11] of the famous Dutch admiral who in an attempt to vomit ruptured his œsophagus completely across, probably at the seat of a carcinoma. Rosenthal [124a] mentions the case of a boy in which the œsophagus was pierced by a fish bone. Turner (West) mentions a case of rupture of the œsophagus due to vomiting but without death. Widal [216] mentions two cases due to the use of the stomach tube. Hence the case 6 which we have reported belongs to a very rare variety. Also in case 19 there was an ulcer perforating into the œsophagus.

Tuberculosis of the Spine.—The case of Hughes [107] is of great interest since the caries of the vertebra caused the empyema and the pneumothorax. This patient, therefore, had a passage from the mouth to the right iliac fossa through the lung. Biach reviews other cases due to tuberculosis of the bony framework of the chest.

Strangulated diaphragmatic Hernia.—Struppler's [252] case is perhaps unique, the strangulated loop of intestine infecting the pleural cavity and gas produced by the organism, not from perforation.

Idiopathic Pneumothorax.—The secretion of gas during the first decade of the past century attracted considerable attention. It is in fact the belief in the occurrence of these cases that brought pneumothorax into such prominence at that time. The publications of Dalton on the nature of gases showing the intimate relation between liquids and gases and of Mitchell and Graham on diffusion of gases had recently appeared, and these new ideas were welcomed as explaining some cases of pneumothorax. Again, the ideas of the secretions were now changing and it was believed that any secreting surface under abnormal conditions was capable of producing gas. Such conditions were known as pneumatoses (Piorry,[40] Brierre de Boisemont [47]). Rullier [39] considered Itard's cases as susceptible of some such explanation. At any rate the gas was a stranger and was not atmospheric air. Laennec [41] accepted the possibility without question and separated two groups of cases:

(1) In the acute pleurisy at the onset without decomposition of the fluid.

(2) The secretion of an aeriform fluid in the chest.

But Laennec's following statements are not so easy to understand, for he thinks such aeriform fluid is often formed in the body, and since such idiopathic formation of gas is beyond question in the case of the pericardium, joints and arachnoid, why not also in the pleura? In these cases the gas does no harm to the pleura since it is "animalized;" it also could be absorbed more readily than liquid, even in a few hours. Piorry [42] accepted the secretion of gas as an established fact. The clinicians very soon found that the interest in pneumothorax would wane unless "the primary essention form" could be defended; for if this be not recognized pneumothorax was merely that common condition secondary to perforation of the pleura and described long ago by the surgeons, and not even worthy of a name. In defence of Laennec a French student, Venot,[45] takes up the question and

although he admits that the argument in favor of idiopathic pneumothorax is one of analogy, yet he considers it well proven, and relegates Itard's suggestion to "the ancient school." In symptomatic pneumothorax the gas was air, but in this form it was a permanent gas of unknown composition. This subject was for years a favorite one for dispute. In Great Britain, Houghton [65] mentioned it merely out of respect to Laennec; Graves,[67] basing his opinion on the work of Dalton, Mitchell and Graham, defends Laennec. He considers that the body is not air-tight and that gas easily diffuses through its tissues; but Pool [68] takes the opposite side on this point. Williams [77] states that this form may occur as a terminal symptom in some diseases and is comparable to tympanites of the abdomen. It is not found ante-mortem, but he has found it in some cases post-mortem. Saussier reviews the subject at length and reports a case which he considers confirms Laennec's view. Marais,[90] a student proud of his knowledge of the new physics of that day, states in a rather sophomorical manner that to deny the possibility of the production of enough gas to fill the pleura from two or three ounces of fluid is to ignore one of the best established and recognized laws of physics. To him secretion evidently belonged also to the old school. But he states one thing which is well worthy of consideration, that gas contracts on cooling and that all the air which rushes out of the chest on the autopsy table cannot be that which was present before death, but some must be due to post-mortem changes. Grisolle [91] considered pneumothorax and pneumatosis and thought there was no doubt that the inflamed pleura could secrete gas. Proust,[124] in 1862, denied the possibility of idiopathic pneumothorax and his thesis had great influence in turning the current of opinion against this form. The question, therefore, took another turn and Demarquay [129] first suggested that the gases of the blood could be the source of the gas of pneumothorax; Anstie [140] still held, however, the belief in the origin by secretion, and Demaroux,[104] in 1884, reported the last case attributed to secretion. Many cases belonging to this group will be considered under pneumothorax following paracentesis.

Putrefaction.—The first writer to suggest this as a possible cause of pneumothorax was Benjamin Bell.[34] Itard [36] seems inclined to explain his cases in this way, but distinctly states that he does not care to theorize concerning the origin of the gas. Laennec [41] gives the putrefaction of an effusion the first place in his catalogue of causes and pronounces it "pretty common." The next, decomposition of blood, would also belong in this group, but the last two men considered putrefaction as the causative factor in these cases. As early as 1824 Venot, one of Laennec's admirers, relegated this explanation to the "ancient school," applicable alone to cases of gangrene. For him abnormal secretion was all-sufficient. Forbes,[64] the translator of Laennec's book, in 1834, considered this cause very problematical and suspected an undiscovered fistula. Houghton [65] was very positive in his denial and explains the odor in the reported cases as due to the gangrene of the pleura. Belief in this form gradually died out. Anstie still held it in 1871, but clinicians, as a rule, denied its possibility until Senator,[242] in 1892, reinstated it.

Pneumothorax due to a Gas-producing Organism.—Such cases are the logical successors of the above group. Senator, as mentioned, indicated their possibility. One of the first to be reported is that of Levy.[203] This was the case of a man tapped for pleurisy five times; the organism finally found was, he says, Fraenkel's organism. Widal considered his case due to proteus vulgaris. May and Gebhard's [302] case is one of the best reported. This was a case of hæmothorax following stab wound, which later became infected by the pus organisms and then developed pneumothorax, due to a gas-producing organism, which he states was not Fraenkel's. Hamilton's case [310] was due to the bacillus aerogenes capsulatus, and Finley's [319] to the colon bacillus. It is to be noted that in all of the above cases the chest had been aspirated and credit of infecting the chest may then perhaps belong to those performing the paracentesis.

Crawford,[93] in 1848, reported a case to us very interesting since the pneumothorax was pure, no disease of the lung was demonstrated, and the condition followed fifteen days after a bad infection of the arm. The gas he ascribed to idiopathic secretion. Jochmann [351] reports another very rare case of pneumothorax acutissimus, due to a strangulated diaphragmatic hernia without perforation.

VALVULAR PNEUMOTHORAX.—The first to recognize this form was Davy, who demonstrated in a case with extreme distension of the chest and perforation, a valvular structure which received very careful description. The best early clinical description of such a case is that of Johnson,[56] and his proof of its valvular nature was conclusive. Houghton described the anatomical findings in such cases and stated that the majority of all cases of pneumothorax are valvular. He described the consequences of the valve, among which dilatation of the side is a prominent one. Skoda considered this the type of a fresh pneumothorax and stated that he had never seen a recent case with a truly open fistula. By open fistula, however, was meant one in which the air has free exit as well as entrance to the pleural cavity. Powell [136] made the first determination of a positive pressure in a chest in such a case (Bard) and recommended this procedure for diagnostic purposes, stating that it had been done in a few cases. It is Weil, however, to whom the credit of emphasizing this group of cases is usually given, although Unverricht (Krebs [228]) claims the honor of priority. Unverricht [266] proposed a novel test to determine whether or not the fistula was open. By open, however, he meant that air could enter the chest. The test was the so-called Wasserpfeifengerausch produced by aspiration in case the gas entered the chest in bubbles through a fistula submerged under the surface of the fluid. Riegel [169] heard this same sound during the development of a pneumothorax and not due to aspiration. He considers it a symptom of open fistula. A case showing the valvular, hence the pumping action of the chest, was that of Williams,[231] whose patient insisted on continuous tapping for four and a half days. It was Duncan [70] who first showed that it was the course of the fistula which could give it its valvular nature without the presence of any true anatomical valve. Bouveret [211] is usually accred-

ited with this discovery. It was he though who first insisted that it was not the inspiratory efforts but the expiratory efforts, especially those on coughing, which raise the pressure in the pleural cavity. It was he, said Bard, who first rejected Weil's inspiratory theory. Duplant [325] emphasizes the importance of the inertness of the diaphragm to allow the distension of the chest.

Other theses treating more fully of this group of cases are those of Desbiez,[323] who considers the only convincing symptom is a positive pressure and that to produce this cough is very important; Béclère,[324] who takes much the same view; and Aron.[325] All these, therefore, consider that valvular pneumothorax is a distinctly unfavorable form. Ewald [155] showed that all the signs of a high pressure might be present and yet the fistula be found open at the autopsy. It is Bard [341] who takes a radically opposite view. According to him valvular pneumothorax is a wise provision of nature allowing the closure and obliteration of the fistula, and a condition to be encouraged in its production rather than combatted.

CLOSED PNEUMOTHORAX.—By closed pneumothorax is meant one in which the fistula does not allow the exit or entrance of air. This closure, as Weil [165] pointed out, may be either mechanical, due to the collapse or the compression of the lung, or organic, due to the obliteration of the fistula. This is a necessary process in the cure of any case of pneumothorax.

The group of symptoms which Widal [210] gives for such cases is, immobility of the chest, great distension, dyspnœa with metallic sounds less marked than in the open form, amphoric respiration diminished or absent, and considerable displacement of the neighboring organs. This description, however, will apply only to a case of valvular pneumothorax which has just become closed, leaving the chest under high intrapleural pressure.

OPEN PNEUMOTHORAX.—By open pneumothorax is meant either one in which air is allowed to enter the chest, or one in which the air may enter and leave the pleura. Cases in which the air can enter but not leave belong to the group of valvular pneumothorax and hence need not be considered here. Skoda [81] stated that he had never seen this form in a recent case, but that it was only in rare, long-standing cases that an open fistula arises through ulceration. Bard has shown that the nature of the lung tissue explains a valvular pneumothorax, a fact which was formerly recognized by Lister, and that the fistula could be considered open only in case it was large and with walls rigid enough to prevent its collapse. Such a condition would be present in a very long-standing case. The signs which Riegel [189] has assigned to this form are, some dilatation and displacement, (symptoms not due to positive intrapleural pressure and called by Unverricht [163] retraction phenomena), rapid changes in the pressure, Wintrich's change of pitch on percussion with the mouth open and closed, "maulvolle" expectoration, gas analysis showing a percentage of carbon dioxide less than 5 per cent, and a rapid return to atmospheric pressure after aspi-

ration. Riegel, however, had in mind probably the valvular pneumothorax. The symptoms which Widal gives include metallic respiration at a maximum, displacement of the organs at a minimum with amphoric breath sounds and metallic tinkling. Szupak [239] added long duration as a symptom of an open fistula.

PNEUMOTHORAX ACUTISSIMUS.—This is the name which Unverricht [268] gave to those cases which die during the first day after the onset. It is the same form which Bouveret [211] called "suffocative pneumothorax." Benjamin Bell [34] was the first to call attention to this form. He says that the symptoms arise more rapidly to an alarming height than in the case of a fluid exudate, and that the patient may die in a few hours from pneumothorax due to a fractured rib. Among the many interesting cases are those of Desplats [158]; Dolosea,[103] whose patient died in three hours, a case due to empyema; Monneret,[194] a case due to acute pneumonia, followed by gangrene and death within 24 hours; Struppler,[352] with death in ten hours, the immediate cause not known, but the condition following an incarcerated diaphragmatic hernia without perforation. Bouveret [211] reported two cases; Williams [55] described these cases as rare, also Stokes.[70] Galliard [343] reported a case in a woman who had been previously healthy. The suffocation was extreme, respirations being 64 to the minute, but this was saved by pleurotomy. Our Cases 9 and 10 belong to this group.

DOUBLE PNEUMOTHORAX.—Laennec states that frequently at autopsy had he noticed an escape of gas from both pleural cavities. The explanation of this we will try to give at another point. He attributed it to post-mortem changes, but he reported two genuine cases. Natrop [179] in 1882 reviewed the cases that he could collect, seven in number, and reported one with entire pneumothorax on the right side, and encapsulated on the left. Lasius [234] selected this as a topic of his thesis and reported one case. Fraenkel's case is very important. The pneumothorax on the left side was produced by an empyema perforating through the lung. The tuberculosis then perforated the mediastinum, causing right-sided pneumothorax. The patient lived 1¾ hours, respiring with a zone of normal tissue which the tuberculous consolidation prevented from collapse. Drasche [316] reported 3 cases, one of whom died at once, one in 2, and the third in 12 hrs.

ACTIVE AND PASSIVE PNEUMOTHORAX.—These are names given by Williams [55] to the two clinical types into which he divides all cases. In the one, the active, there are signs of positive intrapleural pressure; in the second the air merely fills the space left by the absorption of an exudate which leaves a space that the lung cannot expand to fill. This is really the classification which Itard made. To us the active is synonymous with the valvular, but to these men it had a very different meaning, Williams calling passive pneumothorax also the pneumothorax of necessity; the active he pronounced very rare.

PYOPNEUMOTHORAX NECESSITATIS.—Very few cases of this description have been published. In a case of Stokes' [70] the pus traveled under the skin to the scrotum. In Galliard's [260] the remarkable condition

was present of the pus in the lumbar region and the chest contained only air. It was he [278] who gave the name. Of our cases, No. 6, No. 41 and No. 16 may be added.

PNEUMOTHORAX IN CHILDREN.—The first of these cases was reported in 1843 by Lees [86] and was due to the rupture of an adhesion in a tuberculous child two years old. This was the youngest case to date. Pollock's case was due to tuberculosis. Ruge's [156] was a case of a new-born who died during the first day, evidently from pneumothorax following emphysema due to violent crying. Cnopf [252] reviews all of the literature on the question and reports four new cases. His article contains Lentz's classification of 35 cases, and Steffen's review of 16 cases. Zuppinger's [353] paper is also valuable. Leichtenstern [160] discusses the etiology well. Jadelot [88] reports the case of a girl nine years old. Pollock [125] of a child three and a half years old. Nichols [224] states that the course is more rapid in the child than in the adult. Cnopf states that the disease due to neighboring organs is rare in children. The reason rupture of the lung is more common in children than adults is ascribed by Demarquay [129] to the relatively greater strength of their respiratory muscles.

RECURRENT PNEUMOTHORAX.—The first case is that of Gabb's,[219] in which there were probably four recurrences on the same side and without known cause. West [225] reported a case with probably three attacks and yet very few symptoms. Among the cases with one recurrence are those of Knight,[232] Finney.[301] This latter case was that of an 18-year-old man, without strain, with very few symptoms and with an interval between the attacks of about two weeks. Rendu [335] stated that one of his cases of pneumothorax following aspiration was clearly the recurrence of an old pneumothorax. Maillart and Lasserre [357] report that 7 of 51 spontaneous cases recurred. Weber [332] says that his father had a case in which the interval between the two attacks was two years. It is of interest that so many of these cases should have been accompanied by so few symptoms; also that such a large percentage of the spontaneous cases should be cases with recurrence. It would seem to be true that such persons had "weak lungs," either an acquired or an inherited characteristic. One case of particular importance is that of Goodhard,[265] in which the first attack was on one side, the recurrence on the other and both with very few symptoms.

We have no case of spontaneous recurrent pneumothorax, but attention may be called to Case 22.

CHAPTER IV.

The Mechanics of Pneumothorax.

The thorax is mechanically but a closed chamber enclosing the lungs and other intrathoracic organs. The only external opening, however, communicates with the interior of the lungs. Of the respiratory movements, inspiration consists of an increase in size of the

thoracic cavity, brought about by the movements of the ribs and of the diaphragm, and to a much less extent by the contraction of the heart. Expiration, for the most part passive and due to the fall of the ribs, the elastic recoil of the chest and abdominal walls, is a reduction in the size of this cavity.

Since the interior of the lungs is in free communication with the external air it follows that air entering the trachea will, by stretching these, force them to fill the increase in space made by the respiratory movements.

The lungs even at their minimal volume at the end of deepest expiration are still stretched beyond the volume they would assume if the thorax were opened, hence even then there obtains a negative pressure in the potential pleural cavity. This negative tension becomes greater in almost direct proportion to the depth of the inspiratory act.

This *normal negative tension* has long interested men, and its cause, amount and significance have been a subject of frequent investigation. One of the best reviews of the subject is that of Heynsius (*Pflüger's Archiv,* 1882). He says that Donders first showed the importance of the elasticity of the lung for venous circulation. Donders, as a result of observations made with a manometer inserted in the trachea of bodies before the chest is opened, and of experiments on living animals, decided that in man the intrapleural tension at the end of quiet expiration was —7½ mm. Hg., of inspiration —9 mm. Hg., and that of this three-fourths was due to the elasticity of the lung, the remaining one-fourth to its tonicity, which latter disappears about fifteen minutes after death. This opinion obtained for about thirty years, when Gerlach showed tonicity to be unimportant. But Donders proved his point; there is a negative pressure in the chest, greatest during inspiration, which has an aspirating effect on the venous circulation.

Jäger, in his work on pulmonary circulation (*Pflüger's Archiv,* 1879), showed the need of more accurate figures, and since of a dead lung the elasticity changes with each distension, only living or perfectly fresh lungs should be studied. Hutchinson, working with two lungs immediately after death, found their elastic recoil to be 4.5 mm. Hg. He next ried to measure it in living animals by putting a canula in the chest, but such results are inaccurate since the respiration remains no longer normal. Adamkiewicz and Jacobson inserted a

canula into the pericardium and found during normal respiration a tension of —3 to —5 mm. Hg. Heger and Pehl used this method. Rosenthal and others determined the tension in the œsophagus; a bad method. d'Arsonval and Frederick inserted the canula into the bared pleura, and found —4.4 mm. Hg. Heynsius abandoned all direct methods and uses one which he thinks above criticism, that of determining tension in the trachea of the opened thorax. To determine that at inspiration the lungs are filled by the aspiration produced by pulling down the diaphragm. His work is very careful. His results are that for dogs, inspiration = —7.5; expiration —4 mm. Hg. For rabbits —6.3 and —2.4 mm. Hg. respectively. He finds the tonus may be disregarded, also the effect of the blood tension on the elasticity may be disregarded. (d'Arsonval considered this an important item.) He then criticises the former observers' work who obtained results which were too low, and remarks that those dogs give largest values which have not been used for hard work. For men he will accept Hutchinson's figures until better are obtained.

Violent inspiratory efforts will increase the negative tension even to —30 mm. and violent expiratory efforts even to + 80 to 100 mm. Hg.

The *pressures in the trachea* are to us of considerable interest since upon them does the aeration of a collapsed lung in pneumothorax depend. These figures, according to Landois are, on inspiration — 1, on expiration + 2 to + 3 mm. Hg., the average + 0.5 to 1 mm. Hg.; according to Morat and Doyon, on inspiration — 3, on expiration + 2, with an average of — 0.5 mm. Hg. (quoted from Bard[341]). West[276] gives them as —0.5 on inspiration, + 2 to + 3 mm. Hg. on expiration. In considering the respiration phenomena in pneumothorax this intratracheal oscillation must be considered.

The intrapleural tension is, said West,[276] the result of the rigidity of the chest wall, the elasticity of the lung and the movements of respiration.

Perls (*Arch. f. klin. Med.*, 1869) mentions the very early work of Carson (*Phil. Trans.*, 1819) on the *elasticity of the lungs* of animals, and reviews Donders' work at length, which Perls continues with reference to pathological lungs in one hundred cases, a canula being inserted in the trachea. He makes one point which has been repeated and reiterated several times, that the collapse

of the lung on opening the thorax is apparent as well as real, since on removing the sternum the chest wall widens, the cut ends of the ribs spring apart. We do not know how these writers performed their autopsies, but using the technic followed in this hospital, removing the sternum before any of the abdominal organs, this is not true, for then the cut ends of the ribs even are closer than before, due perhaps to the falling forward of the liver and the consequent tension on the diaphragm.

This writer's conclusions are of considerable interest to one working on that point. For us its chief interest lies in his reference to the effects of arsenic poisoning, typhus and typhoid fever on elasticity, which conditions seem to diminish it. He suggests that œdema of the lung, hypostatic congestion, etc., so common in such diseases, are the cause.

An illustration of loss of elasticity of the lung due to arsenic poisoning to which Perle refers was that Grohe and Mosler (*Virchow's Arch.*, 1865). This was the case of a child 2 years old who died seventeen hours after a toxic dose of arsenic. At autopsy was found acute atelectasis of the surface of the lungs, due to the efforts of vomiting, while the rest was air-containing. They considered present a loss of elasticity of the pulmonary tissue due to the direct action of the arsenic.

That the bronchi have broncho-dilator and broncho-constrictor nerve fibers, see Doyon (*Arch. d. Phys.*, IX, 1897, p. 412).

Granted then that tonicity is of no importance and that arterial tension has no influence on the elastic tension of the lung, we have remaining but the elasticity of the pulmonary tissue due doubtless to the abundance of its elastic fibers. This in the case of a normal lung will produce a diminution to about one-half (West) of the size which the lung had before opening the chest. West,[276] however, believed the pulmonary tone to be important and spoke at length of the "acute pulmonary tympanites" seen in disease and over a pulmonic area, in which case there is no collapse of the lung yet a Skodaic percussion note.

Of the diminished elasticity in certain fevers we cannot speak except to mention one case which we hoped would throw some light on the question. It was the case of a man who had run an especially severe course of typhoid fever, without pulmonary features, which with a relapse meant fever of over one hundred days. He died in a

very weak condition. At the autopsy unfortunately the pressure was not measured, but the faint inrush of air on carefully opening the pleura was listened for and heard, and the lung collapsed to about the usual size; a true collapse, not an apparent one.

As to the *amount* of the *negative tension,* Hutchinson's calculation of 4.5 mm. Hg. has been mentioned. Donders', however, are the figures which Weil [172] mentions. The only direct observation on a normal living person is that of Aron.[358] This person in normal condition allowed several observations to be made. The average of thirty-six readings made during quiet respiration was — 4.65 mm. Hg. and — 3.02 mm. Hg. at the height of expiration. The maximum reading for quiet inspirations was — 5.09 mm., the minimal — 4.23 mm., the minimal expiratory pressure — 2.54 mm. Hg. (these pressures are calculated from those read on a glycerine manometer).

In a case of healing empyema the pressures on quiet inspiration varied from — 4.5 mm. to — 6.85, the expiration from — 1.9 mm. to — 3.9 mm. Hg. Many readings made during different postures, sitting, lying, were also made, which may be found in the abstract. These figures cannot of course compare for value with those of the normal person.

This elastic tension producing a negative intrapleural pressure is of great moment in the organism, although the body can adapt itself to great fluctuations in it. Normally it aids the venous flow to the heart. This was beautifully seen in a case of splenomyelogenous leukæmia recently demonstrated by Dr. Osler. The veins of the forearm were full; with each expiration they distended, with each inspiration collapsed. The intercostal spaces are apparent because the soft tissues are pushed inward by a relative positive external pressure which is equal to the negative intrapleural pressure. The liver, because of this intrapleural negative tension, exerts only a part of its weight on the abdominal organs, for this tension will hold up against gravity the diaphragm and a layer of liver about 5 cm. thick. The heart also lies balanced between the two pleural cavities.

When the capacity of the thoracic cavity is encroached upon in any way, whether by fluid or gas or for any other reason, the lungs having less space to fill will contract through their elasticity; the negative tension therefore in the chest is diminished, since its degree varies with the amount of distension of the lung. The intercostal spaces are not so much "drawn" in, hence are less evident;

the liver is no longer partially supported and takes a lower position, and the heart, the most delicate index of a change of tension on either side, will move toward the sound side, pushed in that direction by a force equal to the difference of those in the two pleural cavities. The term used by Powell, Garland and others, "drawn over to the good side by the negative pressure," is unfortunate since there is no drawing done unless pleural adhesions be present, but it emphasized the point desired by these writers, that these dislocations are not necessarily due to a positive pressure, that is, one above the atmospheric. As soon, however, as the elastic recoil of the lungs has been entirely spent an increase in the fluid or air will cause truly pressure symptoms, although we shall try to show that even then the organism will by enlarging the capacity of the chest or by paralysis hence flaccidity of its walls try to prevent true pressure symptoms, or at least try to keep the pressure so low that the inspiratory movement will still produce a fair intrapleural pressure oscillation, hence allowing the lung to expand.

But a loss of elasticity of lung tissue will have much the same result as an effusion. We well remember one case which the pathologist was demonstrating to his class. "This case has fluid in each chest," said he, to the embarrassment of the clinicians whose patient it has been, when he opened the abdomen, found the liver quite below the costal margin and the diaphragm low on both sides. He opened the chest. "There is no fluid here," he added hastily, "but the position of the diaphragm may be easily explained; it was pushed down by these very emphysematous lungs." But was it "pushed down"? The elasticity of an emphysematous lung is notoriously diminished and the liver was not so well held up; also perhaps the organism dictated that position that the respiratory movements might be more effectual.

In case there is a wound through the visceral or parietal pleura which puts the pleural cavity in communication with a gas or air-containing cavity or with the outside, as a rule the lung collapses through its elastic tension, and air enters. This was first shown by Hillis and Lower, two English physicians at the end of the seventeenth century (Nichols [224]). But does this always occur? It was the cases of hernia of the lung (Boyer [38]) which made earlier writers doubt the elasticity of the lung, and Bromfield and Hewson speak of the value

of a large excision that the air may freely escape and the lungs be given plenty of room to expand. In case the cut in the chest wall be large enough and the lung relatively little injured, the lung will be protruded through the hole with such force that the opening must be enlarged before the lung can be pushed back.

In other cases with large chest wounds the lung does not collapse. West [209] mentions two such cases. In one the wound was three inches long; in another the lung was torn by a fractured rib over an area two inches square, yet in neither case was pneumothorax the result, and adhesions were not present to prevent its collapse.

Auquier reviews the matter as follows: It was van Swieten, said he, who first said it was the vacuum in the pleura which keeps the lung distended; this was also taught by Bérart and Richet. But, said Auquier, this is not the case; there is no inherent tendency in the chest to the production of pneumothorax, for Dolbeau and Smith in 1868 first likened the sheets of the pleura to two plates of wet glass which will glide easily the one on the other, yet can with difficulty be pulled apart; and Duplay also taught the presence of this physical cohesion between the pleuræ.

West [209] also believed in this cohesion between the pleuræ, which he considered was equivalent to 12.5 mm. Hg., hence the failure of pneumothorax unless the lung is forced away from the chest is easily understood.

Piqué [212] states that Dolbeau found that an incision 1 cm. could be made into the pleura without collapse of the lung. Belief in this cohesion was also held by many later writers.

Smith[175] is said to have shown the importance of the position of the fistula, that it be near the border of a lobe, and Auquier elaborates this view at length.

But there was also much evidence against the existence of such a force. Northrup [228] found no evidence of it, for he carefully opened the pleura by a pin hole and found that the lung collapsed at once, and others came to the same conclusion.

The experiments we have made on this point showed no such cohesion. If, for instance, there is such a force, and a few cubic centimeters of air be introduced one would expect the pneumothorax to be limited; hence Auquier's [175] classification of pneumothorax as monolobular, bilobular, interlobular, lateral, etc. The above experiment we did repeatedly. Into the chest of a dog just

dying, or just after death in which case a little artificial respiration was used, varying amounts of air were introduced, but always small amounts, less in volume than one lobe of the animal's lung or very much smaller. The animal was then frozen in the cold-storage chamber of the anatomical department. At the end of three days, at which time the dog was solid, the chest was opened and a cast of the air-containing cavity made with plaster of Paris. In each case we found the cavity at the uppermost part of the chest; at the apex if the animal was hung head up; along the spine if left lying on the abdomen.

The physiologists (see *Foster's Physiology*) know nothing of such an cohesion. The surgeons I have asked have seen no evidence of it. I assume from a recent article of West that he does not now insist upon it.

Again, the evidence West[209] presents for such a force is derived largely from experiments with peritoneal surfaces in contact. These we repeated. It is true that sheets of stomach on discs of wood will stick by their peritoneal surfaces, but one point we found and would emphasize, since West should not accept one manifestation of this force and disregard others; although these surfaces did strongly adhere, they did not glide one on the other like plates of wet glass, but when on edge and then weighted it took almost as much weight to make them glide as to pull them apart. Yet the pleural surfaces do normally glide easily and smoothly.

How then are these cases of non-collapse of the lung to be explained? From the earlier writers the expansion of the collapsed lung when the air has free exit from the pleura has been recognized. This can easily be confirmed on dogs (Dwyer[227]) and is beautifully seen on the operating table, I am told by surgeons. By a few forced expirations the dog or person can inflate the collapsed lung by means of the expiratory pressure of the sound lung, which forces the air across into the collapsed lung. If, however, the closure of the glottis be prevented this cannot occur.[227] Granted there is such a force which can keep the pleuræ in contact, it is not hard to understand how the fibrin from a bleeding wound can soon seal the edges.

Smith claimed to be the first to demonstrate the part taken by the good lung, but Auquier[175] reminds him that Marvel-Lavallée (1850) had preceded him.

When, therefore, air is allowed to enter the pleural cavity the lung

begins to collapse. If the perforation be a parietal one and the diameter of the opening be less than that of the bronchus to that lung, the air will enter with each inspiration and during expiration some, but a less amount, be expelled, until the lung at the end of expiration is just collapsed. Then with each succeeding breath the lung will expand somewhat with inspiration, since more air can enter through the bronchus to fill the partial vacuum than through the perforation. With each expiration the lung will assume its former collapsed condition, since the air escapes more easily from the bronchus. The smaller the hole the greater the expansion of the lung, and in case it be then closed the lung can take no small part in respiration. This is essentially the description of events given by John Bell,[95] who said the thorax was like a pair of bellows, the lungs passive and moved only by the vacuum. If the opening be not a free one the expiration will force the air into the subcutaneous tissue. If a very strong blast of air issues from the wound it is proof, said he, that the lung is not injured. (See also Rumpf,[96] who mentioned the part the collapsed lung could play in respiration.)

Garland[157] considered that the air is held at the base at first if allowed to enter there, and that the lung as it collapses remains in contact with the pleura until the base has ascended several ribs. In the case of effusions some (Cohnheim) taught that the lower lobe became airless first, but others agree that the lung collapses as a whole (Calvert, *St. Barth. Hosp. Rep.,* 1892).

The importance of the condition of the mediastinum was emphasized by Sehrwald,[222] for if it be pathologically stiffened the good lung is much less affected and collapses less than when normal, hence the worst effects of open pneumothorax (external) are seen in normal persons. Although in quiet respiration the central tendon of the diaphragm does not stir, hence the mediastinum is quiet, and the diaphragm by assuming a more conical shape increases the capacity of the cavity, yet in forced respiration the mediastinum is put on the stretch, thus aiding the respiration of the unaffected side.

That the lung in pneumothorax does not entirely collapse and still takes part in respiration, and does not alone apparently expand as Reineboth[282] thought, was proven by the experiments of Hellin[348] on double pneumothorax.

What the pleural volume is, that is, the amount of air which will enter before the intrapleural tension reaches zero, has in man not

been directly determined. This is a matter of much interest; it shows the point at which pressure becomes really positive. Yet this can not be a fixed amount. The lung first collapses and then in many cases becomes atelectatic, thus increasing the pleural volume; again, the dislocation of the diaphragm increases the cavity without affecting the pressure. Weil decided as a result of animal experiments that in man the volume might be from 1800 to 2400 cc., and mentioned the fact, which we in several cases could confirm, that when that much fluid is in the chest the pressure will not be above zero.

The above remarks have concerned only open pneumothorax, that is, one with an open external wound. In case the wound be through the lung quite a different series of changes obtains, for a fresh wound, even though large, is still valvular. This was shown by Castelnau (Marais [90]), mentioned again by Lister (Johnson [182]), but the idea elaborated especially by Bard.[341]

These cases of *valvular pneumothorax* may be divided into two groups. First are the rare suffocative cases emphasized by Unterricht, in which the dyspnœa and distension increase until death unless operation be performed; and second those cases, which form the majority of all, which soon reach a condition of readjustment and then, although the pressure may remain positive, the patient enjoy a comparative freedom from symptoms.

Experimentally these cases were first studied by Weil,[165] who used, however, an external valvular fistula; at this point we will review the work done on animals, including open and closed pneumothorax.

Weil recognized three forms of cases, the open, in which the air had as free exit as entrance; the valvular, in which its entrance was more free; and the closed, in which it had neither. The latter may be organically closed or mechanically from the compression of the lung. The manometer was said (Weil [165]) to be the best way of determining these forms, since in closed the mean pressure is negative; in valvular, positive; in open, zero. Seifert [185] reported two cases at which on the autopsy table by the manometer the diagnosis of open pneumothorax was made and confirmed anatomically. At this point we would insist that pressure after death is absolutely no index of that during life, and that anatomically the diagnosis of an open fistula is extremely hazardous.

In the valvular form air enters and since it does not, or not as rapidly, escape, accumulates, causing the positive tension which is said to produce that important group of so-called pressure symptoms. Weil considered that it was during inspiration that air enters. If so alone the tension at the height of inspiration could never be positive.

It was Bouveret [211] who said that Weil was incorrect and that really it was the violent expiratory efforts of coughing, e. g. that forced air into the pleura.

Bard [341] reviews the question at length. In a long series of animal experiments he demonstrates anew that the lung tissue is valvular, even when the pulmonary wound is great, and that coughing really forces air from the chest instead of into it. He considers that owing to a beneficial adaptation the organism produces a positive pressure of + 7 to + 8 mm. Hg., which is of decided advantage to the animal in the closure of the fistula; that this positive tension is produced by the regular play of the respiratory movements, and equals the tension of a normal lung. And so it would if Donders' figures were correct, but not if Aron's are better ones. He thinks that no case of pure pneumothorax is on record with a tension proven above this; and since the animal suffers when this is raised, we presume he would thus explain the dyspnœa of a man in whose chest was a greater tension, for many cases of hydro- and pyopneumothorax have a higher pressure. The suffocative cases he does not mention, perhaps since the pressures in none were measured.

Pressures Observed.—Powell [136] was the first to measure the intrapleural tension in pneumothorax during life. In a case of pneumothorax anatomically valvular he found 4 inches H_2O (7.4 mm. Hg.). Later [138] he reported the pressures found in eight cases at autopsy with a maximum of $5\frac{3}{10}$ inches H_2O (= about 10 mm. Hg.). Weil [172] later reported the pressures at autopsy of six tuberculous cases; all were positive, from 3 to 14 cm. H_2O (2.2 mm. to 10 mm. Hg.), and from the chests from 50 to 560 cc. of gas escaped.

West reported one case with + $2\frac{1}{4}$ in. H_2O at expiration and $\frac{1}{2}$ in. H_2O at inspiration, and another with a mean pressure of 25 cm. H_2O (18.4 mm. Hg.), and with at maximum expiration 33.5 cm., minimum inspiration 17.25 cm. H_2O. The gas in the case contained 12 per cent CO_2.

Weil [201] adds seven more cases all with positive pressures varying from + 2 to + 10 mm. Hg., and from which 10 to 720 cc. of air

escaped. In one case he gives several determinations made at different times to show the changes as the patient improved.

West [257] in a case of pleural effusion before aspiration found a pressure of —4 in. H_2O with a respiratory oscillation of $\frac{1}{2}$ in. The aspiration produced pneumothorax and the pressure became + $4\frac{1}{2}$ in. H_2O with an oscillation of $1\frac{1}{2}$ in.

Meunier [262] reports three good cases, illustrating the three forms of pneumothorax with pressure measurements and CO_2 determinations; Lorrain,[267] two cases of valvular pneumothorax.

West [276] measured the tension in twenty cases, and found a maximum of 9 in. H_2O (17 mm. Hg.); he remarks that it is surprising one finds no higher tension. In two cases the pressure was 0, although fluid was present. He concludes that as fluid collects air is absorbed, and that if pressure remains high it is evidence of disease of the pleura, which prevents the absorption of gas.

Aron [258] reports three cases very carefully, one illustrating each group, and confirming Weil's view.

The physical factors in a cure of pneumothorax have especially interested the surgeons. Aufrecht [258] considered that the lung is re-expanded when the external orifice becomes of less diameter than the bronchus, and that adhesions hold the lung. But the whole matter is well discussed by Reineboth,[292] to the abstract of whose paper the reader is referred.

The EFFECT ON THE OTHER LUNG is always appreciable. Unless the mediastinum is pathologically thickened it will be pushed to the sound side, thus limiting respiratory capacity of the good lung. This compression is sometimes evidenced by the atelectatic areas present on this lung.

The RESPIRATORY PHENOMENA at the onset of a pneumothorax were studied by Gilbert and Roger.[237] The respiratory rhythm in dogs was at once much disturbed, the marked inspiratory efforts lasting for a brief time. After the condition was well established in open pneumothorax the rate became faster, the inspiration deeper. In closed, the rate was slower but inspiration slightly deeper, although less so than in the open. In a closed double case the rate increased, the amplitude diminished, and if then one side be opened the amplitude increased.

Krebs [238] found that in open cases the excursions increased in rapidity and depth, and the increase varied directly with the diameter of the fistula so far as rate was concerned, but less for amplitude. He studied the changes also occurring as one type is changed to another.

Blumenthal [241] found the rate increased in all forms; the volume respired per 1 min. was increased in closed and diminished in the open, the diminution varying inversely as the diameter of the fistula; the depth, or amount per respiratory movement, was diminished in all forms, in the open according to the size of the fistula.

Sackur [279] found that immediately after the formation of an open pneumothorax the air respired per minute is just above normal, the increased rate enabling one lung to do the work of both, and the one lung with each respiration does more than one-half the work of two. The mechanism controlling this is, he says, neither the arterial tension nor the vagus nerve, but the diminished O-content of the blood.

Aron's [358] experiments were especially good. The result of a partially forming (valvular) pneumothorax is more frequent and superficial respirations, the chest expanding more forcibly, the diaphragm descending. The breathing movements then increase to a maximum in depth. In closed cases, on the sound side the tension becomes even more negative, the movements are deeper and slower.

The Circulatory Phenomena.—At the onset Gilbert and Roger [237] found in dogs a slight drop or no change at all. Aron took exception to this and was confirmed by Hnatek,[306] who found at the onset a slight rise. This consisted in a temporary rise due to the skin incision, the introduction of the canula, etc.; and a permanent one due to the low O-content of the blood, the dyspnœa, but also to the presence of air in the pleura which caused reflex stimulation through the vagus.

The Valsalva procedure to test the unexpansibility of the lung (Reineboth [326]) makes use of the venous stasis and consequent change in pulse produced by the forced expiratory effort.

Aron [358] found in valvular pneumothorax a rise in pressure even of 3.3 cm. Hg., and in the open a rise although less marked, and considers this a necessary consequence of a change of tension of even one side.

Powell [138] stated that collapse of the lung had been taught to have

no effect on the circulation through it, and he confirmed this by experiment; but the completely collapsed lung he found did impede circulation.

Pleural Reflexes.—Gilbert and Roger [237] state that these are seen as a result of injections into a cavity the seat of an old pleurisy, or as the result of an operation; they are transitory as a rule and consist of fainting spells, convulsive attacks, or temporary paralysis. The importance of reflex stimulation of the vasomotor center was shown by Hnatek.[305]

Paralysis of the Muscles of Respiration.—This is often mentioned as a factor in the distension of the side.

Skoda [116] speaking of circumscribed pneumothorax says there is paralysis of the intercostal muscles due to their unequal tension, hence they are during respiration drawn in and blown out.

Duplant [325] considered the paralysis ("inertness") of the diaphragm a necessary condition for the formation of the positive pressure of valvular pneumothorax.

Maillart and Lasserre [357] reviewed the best proof of the paralysis of the diaphragm, the paradoxical respiratory movements, which would seem to put it beyond doubt, and state that Mignon had shown the same for the other muscles of respiration.

THE PHYSICAL SIGNS.

1. Inspection.

On inspection the patient presents much the same features as one with pleural effusion. Laennec especially emphasized the short-comings of this method of examination. And yet, in a well-marked case, the diagnosis could be thus made; the degree of distension is in no other condition so great, the dyspnœa may be severe, the dislocation of the apex beat, even the depression of the liver with the groove at the costal margin may be apparent.

Subcutaneous Emphysema.—This is a common complication of traumatic pneumothorax. The reference to it by Paré [8] may or may not have any connection with this condition. Littré's [9] case is the record case for amount of distension, perhaps Méry's [10] would have

been had he measured its thickness. In Le Dran's[12] case, due to fractured rib, it was four fingers' breadth deep. A case, important because of the fruit its publication bore, was that of Hunter,[23] and in that case the œdema would seem clinically to have been associated with pneumothorax. See also Russell's, Cheston's and Leake's cases, and Huxham's letter. But these men considered the emphysema important and worthy of treatment. Hewson, however, considered it as a protection to the pleural cavity, preventing the formation of pneumothorax, and a process not to be hindered. That in all such cases the œdema was not the result of a wound of the lung, Petit held, but Boyer[38] denied.

The whole subject receives careful discussion by Demarquay.[129] An interesting illustration of this in connection with tuberculous (?) pneumothorax was reported by Coats,[170] a case with subcutaneous emphysema of the right mammary region.

The character of the wound best adapted to produce emphysema was discussed. John Bell[35] considered a slight superficial wound best, and that the air escaping from the lung at inspiration is pumped into the subcutaneous tissue by expiration.

In our traumatic cases it was several times present, and especially in those due to paracentesis. (See cases 42, 43, 44, 46, 35, 38 and 39).

Local Œdema.—Morgagni[18] mentions œdema of the hand and arm of the affected side as a symptom sometimes observed in dropsy of the thorax. Weil mentions œdema of the hand as an early symptom of pneumothorax. Zahn[240] reported a unique case of œdema of the chest wall, due, he said, to increased intrapleural pressure obstructing the lymph flow.

Striæ Atrophicæ.—But few cases are on record in which these were the result of pneumothorax, although they are associated with pleurisy, pneumonia, and like conditions accompanied by distension of the chest. An interesting case was Gimbert's,[203] in which the striæ were on the unaffected side, a fact which he explains by the added work of the good side in breathing for both. Gilbert[207] reported a case and reviews the literature on that subject.

The Distension of the Affected Side.—This sometimes marked feature was early considered of doubtful value. Laennec,[41] who would of course belittle the evidence given by observation, considered it a

frequent source of error, since the affected side would be considered the normal. He had little to say of the ease with which the interspaces may be seen. During recovery he says the affected side may look smaller. Piorry [42] considered the distension of the intercostal spaces of value only in extreme cases. The distension of the side may be by no means an early pressure symptom, for Houghton [65] early pointed out that the displacement of the heart and the depression of the diaphragm precede it. Neither may it always be present, for Johnson's [56] case for instance presented violent dyspnœa relieved by the escape of gas on tapping. On the other hand there may be distension and all the signs of increased pressure, yet the proof on the autopsy table fail.

The distension is greatest in cases of pneumothorax acutissimus without fluid. (M'Dowel's [114] case.)

Gérard [127a] considered that the side looked larger than it would be found to be on mensuration. Bard [241] cannot consider the distension as a pressure symptom, nevertheless the sequence of distension, dyspnœa, tapping, and relief are so frequent that some at least, and these include cases of pure pneumothorax, may be considered as showing a causal relation. Of these cases are those of Davy, Johnson, Hughes,[197] Auquier,[175] Gould,[153] Mackenzie and Ramskill,[141] v. Ziemssen,[189] Williams,[77] and Noble.[142] An interesting case was that of Williams,[231] who insisted that the aspirating pump be kept in constant use for over four days.

Skoda [116] gave as the elements causing a positive pressure, the valve action of the fistula, the expansion of the air when warmed to body temperature, and the accumulation of fluid.

Until the work of Bard, the distension was quite unanimously accepted as a sign of high intrathoracic pressure, and a good argument in favor of this view was the diminution in size and the relief of the dyspnœa after tapping the distended side, during which procedure air could be heard to hiss out. Yet as Bard has shown there is no case of pure pneumothorax on record in which the pressure has been measured and found to exceed 9 mm. of Hg., and this pressure in many cases causes no inconvenience.

In considering the cause of this symptom, the following points must be borne in mind.

1. The extent of the distension.
2. The actual pressure found.
3. Was this measured during life or after death. The displacement of the heart and liver are not alone signs of high intrapleural tension.

Of our cases, in thirteen the side was noted as distended, and in one locally prominent; in eight cases it was smaller than the normal side. The more interesting cases are: case 15, in this the intrapleural pressure was twice measured, once the day before death and each time a positive pressure of about 8 mm. mercury was found. The perforation was valvular, since after the pressure was diminished by coughing out air it returned soon to the previous height. On the autopsy table, however, a negative pressure of 5 mm. Hg. was found. Hence this case falls within the limits set by Bard, although not one of pure pneumothorax; a post-mortem rise of pressure was not determined. During life the chest was very much distended, surpassing the other side by over 6 cm., a remarkable amount, and it looked very distended; the patient was in great dyspnœa and distress. With the removal of some of the air, the patient was at once relieved and slept the whole afternoon, and a similar relief followed the second tapping. After the air was allowed to escape, (the Béclère apparatus was used) the side was much less distended, the intercostal spaces now visible. This patient was distended and uncomfortable with a pressure of only 9 mm. of mercury, less distended and comfortable, when some air was allowed to escape and yet judging from the rapidity with which the pressure after each expulsion of air returned to 6 to 8 cm. of water, after it had been reduced to — 2 cm. H_2O it would not take long to return to the + 8 mm. Hg. At any rate the diminution in size of the chest and the relief were out of proportion to the change in pressure. In this case at the first tapping with much less fluid (slight movable dulness in the axilla) the intrapleural tension was very similar or slightly greater than with much more fluid.

In case 13 the side was distended yet the pressure was only + 4 mm. Hg. Following the removal of 500 cc. of air the patient was relieved.

In cases 6, 7, 27, the side was distended and the fistula anatomically probably was a valvular one. In cases 3 and 12, so far as could be judged the fistula was open and the side not distended. (No specific note is given of this in case 12). Anatomically, in case 10, and also from the failure of the candle test in case 11, one might believe these cases to have an open perforation, yet during life the side looked distended and measured ½ inch more than the other. But if in case 15 there had been no previous measurements of pressure, the same could have been said.

The reverse was present in cases 11 and perhaps of 6 and 7; during

life there was not enough difference in size to attract attention, yet on the autopsy table gas rushed out.

Of the cases with normal or reduced size of the side we have case 14 (without autopsy). In this case the affected side,(left) measured ½ inch less than the normal, or was about normal. In case 2, mistaken for cavity, the side was flatter. Cases 31 and 22, the one surely, the former probably with the thickened pleuræ, both cases of long-standing trouble, the side was flattened. The same is perhaps true of case 2.

As a case improves, one of the first symptoms is a return to normal of the size of the chest and the depth of the intercostal spaces.

It would seem at first that there was a marked coincidence between the cases with distended side and those in which the lung was found compressed and airless. This is true of cases 7, 15, 1, 6 and 30. Yet the distension in cases 5, 3, 14 and 11 was not enough to warrant any such statement, and cases 4 and 22 have both an airless lung and a contracted side, yet this is not so hard to explain in the latter.

The experimental side of this question is one which has interested us considerably, and we add a few protocols of experiments on dogs.

Our conclusions were the following: The chest of a dog is a cavity capable of considerable variation in size. This increase in size may be brought about by an increased elevation of the ribs, a greater depression of the diaphragm. The movement of the mediastinum is a more passive process. The object of the respiratory movements is to aerate the lungs and the effort of these movements is best measured by the difference in pressure at the end of inspiration and expiration, that is, the oscillation. If the same muscular effort were made when the lungs are collapsed as when normally inflated, we would have a smaller difference in pressure since the collapsed lung would at the end of inspiration exert less elastic tension. To cause the proper aeration therefore the animal must make either faster or deeper respiratory movements. In the case of a dog with closed pneumothorax, we have found the latter to be the method employed. If the capacity of the thorax be encroached upon, by introducing successive portions of either air or fluid, one would expect the negative tension at the end of expiration to progressively diminish to 0 and if the injection be continued to progressively rise in positive value. But this does not happen. To be sure the negative pressure is soon relieved, and if enough be introduced a large positive pressure produced, but the matter is not one of steady progression, the animal is able to accommodate itself to a

considerable encroachment upon the capacity of its chest and still have the pressure nearly 0 at the end of expiration and to produce almost a normal negative tension at inspiration; this it can only do by increasing the capacity of the chest by elevating the ribs or depressing the diaphragm. In this way we may have the chest assume very different sizes and still be of the same intrathoracic tension. But the more the distension necessary to maintain this base line of approximately equal expiratory pressure, the greater the muscular exertion necessary to produce the desired negative inspiratory pressure, and we may have the dog now with a slightly distended chest and breathing quietly, now with a distended chest and very dyspnœic, yet with pressures which differ but little, or at least so little that it is not at all in proportion to the difference in air or fluid injected. The mechanism by which this is attained is probably reflex; the dogs were under an anæsthetic (ether). With each addition of fluid the chest cannot return at the end of expiration to its previous volume, hence accepts that volume as its zero point, and tries to respire as well with this as its starting point.

In these experiments a mercury manometer writing on a drum was used to register the intrapleural tension, although to get the finer variations a Hürthle tambour was used.

January 30, 1903. Dog No. 1.	At end insp.	At end exp.	Difference.
Pressure in chest.	−10	− 4	6
+ 20 cc. Na Cl sol.	− 8	0	8
+ 10 " " " = 30 cc.	− 6	0	6
+ 20 " " " = 50 "	− 6	0	6
+ 20 " " " = 70 "	− 6	0	6
+ 20 " " " = 90 "	− 6	+ 2	8
+ 20 " " " = 110 "	− 6	+ 2	8
+ 20 " " " = 130 "	− 6	+ 2	8
+ 20 " " " = 150 "	− 6	+ 2	8
+ 20 " " " = 170 "	− 4	+ 2	6
+ 20 " " " = 190 "	− 4	+ 2	6
+ 40 " " " = 230 "	− 6	+ 2	8
+ 40 " " " = 270 "	− 2	+ 2	4
+ 50 " " " = 320 "	4	− 2	2
+ 50 " " " = 370 "	− 4	+ 4	8
+ 50 " " " = 420 "	− 2	+ 4	6
+ 50 " " " = 470 "	6	+10	16
Fluid removed.			
− 25 " = 445 "	−10	+14	24
− 50 " = 395 "	− 8	+12	20

(January 30, 1903. Dog No. 1.)		At end insp.	At end exp.	Difference
Fluid removed.				
— 25 cc.	= 370 cc.	—10	+ 6	16
— 50 "	= 320 "	— 8	+ 2	10
— 50 "	= 270 "	—10	0	10
— 50 "	= 220 "	—10	+ 2	12
— 50 "	= 170 "	— 6	0	6
Fluid introduced.				
+ 50 "	= 220 "	— 6	— 2	4
+ 50 "	= 270 "	— 8	0	8
+100 "	= 370 "	— 6	+ 4	10
+100 "	= 470 "	— 4	+ 4	8
+100 "	= 570 "	— 6	+ 6	12
+100 "	= 670 "	— 6	+18	24
Fluid removed.				
—100 "	= 570 "	— 8	+14	22
—100 "	= 470 "	—14	+12	26
—110 "	= 360 "	—12	+ 6	18
—105 "	= 255 "	—10	+ 2	12
— 60 "	= 195 "	—10	— 4	14
Fluid introduced.				
+100 "	= 295 "	— 8	0	8
+100 "	= 395 "	— 8	+ 2	10
+100 "	= 495 "	— 4	+ 4	8
+100 "	= 595 "	— 2	+ 4	6
+100 "	= 695 "	— 4	+ 6	10
+100 "	= 795 "	— 4	+14	16
Fluid removed.				
—100 "	= 695 "	— 6	+10	16
—100 "	= 595 "	— 8	+ 8	16
—100 "	= 495 "	—10	+ 6	16
—100 "	= 395 "	—10	+ 2	12
—100 "	= 295 "	—10	— 6	16

It of course needs no explanation that the figures indicating the residue in the chest really are much too high, since the fluid easily passes into the other pleural cavity and also is rapidly absorbed.

After fluid was introduced the movements were allowed to get quite constant in extent, then the readings made.

Medium sized Dog. No. 2. Amount of physiological salt solution injected into right pleural cavity.	Average pressure at end of insp.	Average at end of exp.	Difference.
	−11 mm. Hg.	− 6 mm. Hg.	5
+ 50 cc.	− 7	− 2	5
+ 50 " = 100	− 7	− 2	5
+ 50 " = 150	− 7	− 2	5
+ 50 " = 200	− 7	− 2	5
+ 50 " = 250	− 6	− 1	5
+ 50 " = 300	− 6	− 1	5
+ 50 " = 350	− 5	− 1	4
+ 50 " = 400	− 6	0	6
+100 " = 500	− 7	+ 2	9
+ 50 " = 550	−10	+ 4	14
+ 50 " = 600	−11	+ 6	17
+100 " = 700	−12	+12	24
Fluid removed.			
−100 " = 600	−12	+ 4	16
−100 " = 500	−11	0	11
−100 " = 400	− 9	− 1	8
−100 " = 300	− 8	− 3	5
− 50 " = 250	−10	− 5	5
− 50 " = 200	−10	− 7	3
− 10 " = 190	− 9	− 3	3
Large Dog.			
June 12, 1899.			
Air introduced			
+71.9 cc. (reduced to 0° 760 mm.)	−22	− 2	20
+76.7 "	−12	0	12
+40 "	−10	0	10
+51 "	− 6	0	6
+98 "	− 4	0	4
June 13.	−20	− 2	18
−28.9 "	− 8	0	8
June 14.	− 7	+ 1	8
−12.7 "	− 9	− 1	8
June 15.	−12	0	12
−31.7 "	−11	0	11
June 16.	−27	+ 5	32
−23.5 "	−13	+ 1	14
July 17.	−10	+ 2	12
−33.4 "	− 9	− 2	7
Exp. 3. Medium sized Dog.			
June 2, 1899.			
Pressure in chest.	−12	−10	2
+30 cc. water.	− 8	− 4	4
+20 " = 50	− 6	− 4	2
+50 " = 100	−10	− 2	8
+30 " = 130	− 8	− 4	4
+40 " = 170	−10	0	10
+60 " = 230	− 8	0	8
+30 " = 260	−10	0	10
+60 " = 320	− 8	0	8
+50 " = 370	− 8	+ 2	10
+40 " = 410	−12	+ 4	16

I think it will be seen from these few tables that the dog can keep its expiratory pressure practically constant or negative, although the amount of fluid in the chest may vary much.

An interesting thing noted when the recording tambour was used is that if the fluid be injected rather rapidly, the first effect is a series of short respirations five or six in number with a very negative pressure on expiration, then the animal takes longer and longer breaths, the lengthening due chiefly, but not entirely, to the depth of expiration until the animal has, as it were, found a range of respiratory movement within which it can comfortably breathe and without too positive an expiratory pressure.

Clinically, somewhat similar processes would seem to occur. The patient's chest varies much in its distension, but evidently not much in its expiratory pressure, never perhaps + 9 mm. Hg. The descent of the diaphragm and the position of the ribs give space enough for the air and fluid but limits the possible play of respiration, hence the dyspnœa. If some of the air or fluid be removed the chest assumes smaller volume, the pressure may not vary much but the patient can breathe more easily because he can make wider movements.

The Position Assumed by the Patient.—While as a rule the patient prefers to lie on the affected side, there are two remarkable cases on record in which the knee-elbow position was chosen. These are those of Morris,[355] and Garde [53] whose patient held that position fifty-six days.

The Position of the Liver.—In right-sided pneumothorax this is a sign of considerable moment since the liver is depressed more than in other conditions, and is lowest in acute cases with only air in the pleura.

In Meckel's case the liver was in the iliac fossa yet only air was present, and much the same could be said of Maillart and Lasserre's [357] case. But this is a common finding. Houghton [65] first called attention to the importance of the sulcus at the costal border in differentiating a depressed from an enlarged liver. In Alexander's [40] case the liver was so twisted that the right lobe was over the spine, and West [164] reported two cases with the notch under the tips of the ninth rib.

Of our right-sided cases the liver was depressed eleven times to a distance of 2 to 10 cm. It is of interest that in cases 1, 17 and 27 in two of which no fluid was present, and in the third (17) with but 425 cc. in an encapsulated cavity, the depression was as great as in cases 3, 5, 11, in which a maximum amount of fluid was probably present.

In left-sided cases, the position of the depressed diaphragm was frequently noted. In case 8 it reached the 7th rib, in cases 3, 6 and 9 it projected below the costal margin, yet none of these cases contained much fluid.

In case 21, a right-sided case, the liver was not palpable.

The Position of the Heart.—It is strange that so striking a feature should have almost escaped Laennec, who merely refers to it. Davy,[44] however, noted it; but to Rayer[53] is due the credit of first emphasizing it. In his two cases so extreme did the displacement seem to him that he considered the question of congenital malformation. Bayle's case, reported by Itard,[56] gave a tympanitic note over the cardiac region.

Houghton[65] first called attention to the fact that it takes less compression of gas to dislocate the heart than to distend the side. This point was carried still further by Skoda (Garland[157]), and Powell,[138] who was the first to measure the tension of the gas and to show that the heart is displaced by a pressure not even atmospheric, and[159] that the heart was the most sensitive sign of a change of pressure. Cotton and Alison[139] attempted to confirm Powell's opinion but we are confident that a pressure determined on the post-mortem table is no index of that which has obtained ante-mortem.

West[188] considered the displacement of the heart one of the best signs of this disease.

In two cases, Banks[110] and Biermer[121] the patient felt the heart move.

Weil[180] considered that a rotation preceded the displacement.

It is not uncommon for the heart to lie entirely to the right of the mid-line in left-sided cases. In right-sided cases it is displaced also, in Harris' case 1½ inches outside of the left mammary line, in Pitt's case 2 in., in Traube's[160a] 3 in.

This, one of the first signs of pneumothorax, is also one of the best indications of a complete return to normal conditions, and the patient is often pronounced cured only when the apex beat has returned to its original position.

In ten of our left-sided cases there was no cardiac dulness at all to the left of the sternum and the heart lay entirely to the right. In cases 4, 18 (a circumscribed case), 31 and 36 the dulness was only diminished.

In the left-sided cases the displacement was extreme in cases 1, 3, 5,

15, 32, and moderate or not present in eight others. The fact that the heart was more vertical than normal was stated or may be suspected in cases 1, 3 and 15.

2. Palpation.

Vocal Fremitus.—This early was seldom emphasized. Often when mentioned it was spoken of as entirely absent. Galliard [213] adds the absence of this to hyperresonance and absence of breath sounds, speaking as if this triad had not before been mentioned. Gérard [127a] speaks of it as more often reduced than absent, and Nothnagel [248] says it is usually present.

Bichat's Sign.—This Piorry [42] introduced into the symptom-complex of pneumothorax as excellent and harmless. This is that pressure on the abdomen is painful when air or fluid is in the chest.

3. Percussion.

Percussion Note.—In pneumothorax Laennec emphasized the deceptive quality of this percussion tone which Itard [30] placed in the symptom-complex, and stated that the full resonance would often mislead the diagnostician, since the normal, and relatively dull, side would be considered the affected side. Perhaps Laennec emphasized the danger too strongly; mediate auscultation was his discovery and any insufficiency in the information given by percussion would increase the value of auscultation. It should, however, be remembered that the methods Laennec used in percussion were not nearly so delicate as those of to-day. Piorry,[54] who introduced mediate percussion, taught the superior value of this to auscultation. Nevertheless the mistake is often made to-day. In reading the notes (dictated during the course of the examination) of physical examinations of the above reported cases, it is not rare to find that the examiner describes the affected side simply as of normal full resonance, describes at length the quality of the relatively dull note of the normal side and not until fremitus is examined and auscultation used does he recognize the affected side.

Piorry's opinion,[54] although interesting, has for us diminished value, since he tried no case of pneumothorax but practiced only on the artificial condition produced at the autopsy table.

It is indeed difficult to discuss the question of tone, for the various observers use various names for the same tone, the same name for various tones.

Skoda described the note as tympanitic, but if the chest be under considerable tension this tympanitic quality will not be heard.

Marais [90] emphasized the value of percussion.

In pure pneumothorax Wintrich considered the tone tympanitic and that by combined auscultation and percussion the size of the cavity could be determined.

Weil quotes Traube as showing that after death the body may give a metallic note not present during life, and this is taken as evidence that the properties of the chest wall have changed, and hence in pneumothorax the abnormal percussion note may depend on abnormal properties of the chest wall. Another evidence of this is the appearance of this metallic quality after aspiration, mentioned by Weil and present in Church's case. Weil made one important point, that it is hyperresonance extending beyond the limits of normal lung resonance which is important.

The question of a dull percussion note is one of particular interest. Saussier [78] and Hayden [339] considered that a thickened pleura could give a dull tone. That a high intrapleural tension could make the note duller believed Skoda,[81 110] Anstie,[140] Hayden,[339] and Hoover.[313] Actual observations are, however, few. Traube [169a] found the tone when the pressure was positive, abnormally loud and deep. Church [151] found that after the gas was allowed to hiss from the chest of his patient the pitch of the tone dropped. Widal [216] said that a high-tension gas gave a high-pitched tone. Hoover [313] said the result was a high-pitched tone of shorter duration. West [209] doubted that the note would become more muffled. Many have doubtless attempted this point on the cadaver and succeeded as well as Hoover,[313] for although his statement is not explicit, the inference is that he failed with the pressures which are possible during life to get a dull tone, hence the distended bladder is a favorite resort and that under high tension gives a dull tone; but that is no fair experiment to apply to the chest. I am sure that on animals a pressure which during life could be borne would give only a more drum-like note. But the matter is a difficult one to discuss. The words full, hyperresonant, deep, tympanitic, et alt., are evidently interchangeably used by many; pitch is seldom distinguished from intensity; to speak in the terms of fluid, we consider that there is no good case on record in which a chest full of air sounded like one full of fluid, although the reverse may be often the case, especially in the young.

The PITCH of the percussion note. Wintrich considered the change of pitch on opening and closing the mouth important in the diagnosis of open pneumothorax. From a change in the pitch could also an increase of fluid be suspected. This Weil was unable to verify.

Traube [160a] made several very interesting observations on the percussion note at autopsy.

Wintrich [111] considered a change in the tone a good index of changes in the amount of air.

Béclère [293] said an increased pressure gave an increased hyperresonance.

Nothnagel [248] said the percussion tone was loud and metallic if the tension was high, but tympanitic if lower.

Piorry [64] found the note more tympanitic if air be forced into the chest.

Saussier said the note was dulled by a thickened pleura.

In thirty-seven of our cases the quality of the tone was described. Of these, twenty-three gave a tone more resonant than normal. These were distributed as follows: tuberculous complete pneumothorax, nine; gangrene of lung, and pulmonary abscess, one each; traumatic, three; pleurisy (following aspiration), nine. Seven cases were described merely as resonant; seven as of defective resonance. The last seven were: total tuberculous, three; circumscribed tuberculous, two; pleurisy, one; amœbic abscess, one.

Granting that the tone of a recent pneumothorax without much increased intrathoracic pressure and with normal pleura would be described as resonant or hyperresonant, we will first consider the cases with dull tones. Case 5 was one of complete tuberculous pneumothorax, and in the last note the tone is described as resonant, rather full pitched. This condition evidently obtained till death, some time later, but since the note is not definitely stated later the case will not be further discussed. In case 14 the relative dulness obtained until death. At autopsy the pleural cavity was large, only five hundred cubic centimeters of pus were present. The gas was present under some pressure, the pleura was thickened. Case 2, no autopsy. The circumscribed cases are easy to understand; the cavities were small, the pleuræ much thickened. In case 23 the pleura was much thickened, the air-containing cavity small. In case 22 the cavity had board-like walls and contained chiefly pus.

THE CONDITION OF THE PLEURA.—It may be coincidence, but in those cases in which emphasis was laid on the thickness of the pleura the note was described as particularly hyperresonant. This was true of cases 6, 7, 15, and especially 1, 3, also 4 and 27. Cases 4 and 7 are exceptions, yet it is not strange that those chests with thickened walls rendered more elastic by a great deal of granulation or fibrous tissue should give a more drum-like tone.

Of course the POSITION OF THE LUNG will modify the tone, and where it lies its presence will be demonstrated by the change in the tone. This was especially well seen in cases 7, 10, 3, 36, 14, 15, and 22. In case 1 the lung was evidently nowhere in contact with the chest wall.

Where the lung comes in contact with the chest wall the note will be found impaired. As a rule, the lung lies along the spine posterior, or when, as usually is the case, there are extensive adhesions at the apex, the impaired resonance may be found over the upper lobe.

In some cases there are marked differences in the percussion tone which are not easily explained. In case 25 the amphoric quality of the note was best made out below the angle of the scapula, although the lung was very small and compressed to the posterior internal part of the chest. In case 14 the note was especially tympanitic in the 3rd, 4th and 5th interspaces inside the mammary line, and more impaired outside. This could not have been explained by the lung's position. In case 38, also, the tympanitic area was below the angle of the scapula. Of course the relative dulness in the scapular region, due to the thickness of the muscles, attached to this bone, must be allowed for.

The CRACKED-POT sound is sometimes described in cases of pneumothorax. This may be due to two things: first, there may be a cavity in the lung large enough and in a sufficiently favorable position to give this sign; second, if the perforation be a large open one the sound may be here obtained. The former probably explains its presence in cases 4 and 2. Very much the same may be said for Wintrich's change of pitch.

THE PERCUSSION TONE OVER FLUID.—This one would expect to be dull but in fact often is not, even when much fluid is present. Stokes [70] noted this point, for he said if the chest was hyperresonant to the base and much fluid be present it was contained in the concavity

of a paralyzed diaphragm which projected downward into the abdomen. Williams [77] thought he could tell the exact level of the fluid by percussion. Skoda's [81] rule is a much quoted one, that to estimate the amount of fluid, double that indicated by percussion. But many since then, Wintrich included, consider their ability in percussion superior to that of this famous physical diagnostician. In this connection Weil mentions Senator's case which was hyperresonant to the twelfth rib behind even after 800 cc. of dilute salicylic acid had been added to the fluid already present.

The reason for this is rather hard to give if the percussion had been lightly performed; the vibrations may be communicated to the air chamber above which determined the quality of the tone. If however, the vibration be hard and on a rib the fact that each rib reaches obliquely downwards and forwards, hence may be more than half against the air space which it will set in vibration, may explain much.

The fact remains that if in pleurisy with effusion one must always be ready to find more fluid than he expected (and Prof. Naunyn would emphasize this point repeatedly to his students), in hydro- or pyopneumothorax one must expect even more. It may be hard to believe this as one percusses the chest, but the humiliating disclosures at the autopsy table in some cases (case 15 e. g.), will teach the young clinician not to claim greater delicacy of percussatory perception than Skoda. We inserted in case 15 a needle over a hyperresonant area of a pneumothorax, expecting to get only gas for analysis and found we were below the level of the fluid.

Movable Dulness.—This as an important sign of pneumothorax was first emphasized by Piorry.[42 54] Laennec [41] mentions it under the diagnosis of pneumothorax with fluid; but he did not believe it valuable in pleurisy with effusion and hence scarcely mentions it in connection with pneumothorax, except in one place and there he says the pleural adhesions will interfere, and he does not think one justified in trying it because of the inconvenience to the patient and physician.

Piorry the inventor of mediate percussion added movable dulness to the symptom-complex as an important element, but had no cases on which to test it. Houghton considered it very valuable.

A marked movable dulness can mean nothing but pneumothorax. It takes however, considerable fluid to allow it to be perceived, for there may be quite a few hundred cubic centimeters of fluid present,

the succussion splash beautiful, and resonance to the base behind. Again with enough fluid the diaphragm may project convex downward, and so much be held in that depression (Finlay and others), that little comes in contact with the ribs, hence the position of the liver is, in right-sided cases, an important point in estimating the amount of fluid.

It is hard to judge the rapidity of effusion, hence the following figures may be considered maximal. In case 4 the movable dulness was of three interspaces, from the 6th rib recumbent to the 3rd rib when erect, and the surgical note is that a moderate amount of pus was found. In case 7, the examiner thought he found slight motion of dulness in the axilla, and only 50 cc. of fluid were found. Case 1: no dulness at all was found and the liver was depressed; eight days later 500 cc. of fluid were present and the liver a handbreadth below the costal margin. In case 3, the movable dulness was "marked," the liver was depressed; about 400 cc. of pus must have been present. With this patient recumbent there was no dulness over the front of the chest. Case 4: here again the dulness was markedly movable, the liver depressed 2½ inches and over 4000 cc. of fluid present. In case 15, no well marked movable dulness was found save in the axilla. The liver was depressed much the day of death, yet over 2000 cc. were present.

In the pleurisy cases the movable dulness is a marked and interesting feature, for in some cases it leads to the diagnosis. In case 30 the dulness moved from the costal margin to the 5th rib; about 2000 cc. of fluid were present: in case 36 from the costal margin to 4th rib, only about 800 cc. could have been present, and yet the lung was compressed to very small volume. Case 37, also 38 are especially interesting for instead of fluid moving in an air chamber we have a bubble of air moving always to the top of a chest almost full of fluid. This bubble in case 37, could be demonstrated on the front of the recumbent patient at the apex when erect, in the axilla when lying on the opposite side. Such a movable dulness can only mean air. In case 39 the dulness was exceedingly movable. In case 21 it moved from the costal margin to the third rib, in case 22, 10 cm. These are the most marked cases of our series.

Claquement costo-hepatique.—This sign proposed by Saussier[78] for cases of the right side, consists in percussion over the eighth rib.

If no fluid be present the rib can be felt to strike the liver. The liver must not be depressed. He does not claim much for this sign, but thinks in some cases [84] it may be of value.

4. Auscultation.

The Amphoric Phenomena.—Under this heading are included the amphoric respiration sounds, the amphoric voice and heart sounds and the metallic tinkling. The coin sound and the amphoric percussion tone are treated under separate headings.

At the onset should be emphasized the difficulty in discussing phenomena which are only audible. They can neither be figured, pictured nor measured, hence objectively they are hard to record so that others may understand what was heard. The same sound is described by different persons with different adjectives. The phenomena of pneumothorax, however, are quite characteristic, that is, are often quite strikingly different from those of other conditions, so that there is considerable uniformity in description of the sounds in well marked cases. In the cases we have above recorded there is, we think, unusual uniformity in the use of descriptive terms, since nearly all the cases were examined by Dr. Osler.

Amphoric Respiration.—As the word indicates, by this is meant breath sounds resembling those produced by blowing into an empty decanter. This sign was first described by Laennec, as "bourdonnement amphorique." Forbes translates this "utricular buzzing" or "amphoric resonance," both of which terms Houghton [85] considered as foreign as the French, hence returned to Laennec's terminology. We shall use the term "amphoric respiration." He explained these sounds by the air entering and leaving the pleural cavity through an open fistula. He considered them related to metallic tinkling, the former being produced at a wide fistula, the latter at a narrow one. Williams [85] held a similar view and mentions an apparent antagonism between these two phenomena, due, he thought, to the disturbance of vibrations. Skoda discussed the subject at length and satisfactorily; he considered the nature of the resonance chamber and of its walls, all important; fluid and an open fistula, superfluous. Amphoric respiration is therefore only intensified tracheal respiration.

Marais [90] considered the tension within the cavity to be important, and by an increase of tension explained the intensification of the mur-

mur at the end of expiration. Another interesting point he emphasizes is the respiratory power of the individual, hence the absence of the signs before death. This may explain the appearance of amphoric respiration on the fourth day of Weber's [332] case, the respiratory power then increasing. The amount of gas and the compression of the lung tissue are also important points. But a fistula is not necessary, it is enough that the cavity can be set into vibration by the vibrations of the good lung. In short he accepts Skoda's idea.

Monneret [194] came to Laennec's aid with three cases which he thought prove the importance of an open fistula, and yet he concluded that amphoric respiration is only tubular in a large chamber and that the fistula need not be open.

Weil [201] whose studies were particularly careful, considered this form of respiration best produced by an open fistula into a pleural cavity with regular smooth walls of a certain thickness and tension, and the sound to be of a certain pitch. In fresh cases it is not present. It did not coincide in intensity with the position of the fistula, but was most often at a maximum in the lower back; yet failure of the amphoric respiration was evidence in favor of the closure of the fistula.

The controversy concerning the patency of the fistula was a most animated one. Garde [53] reported a case with distended side, a drum-like percussion note and remarkable amphoric respiration, yet at autopsy, six days later, while a large perforation was found, air rushed from the chest, hence there is some doubt as to its patency. Corrigan's [127] case was similar.

Yet that the fistula need not be open was admitted as soon as it was found that most cases were really valvular. Lister (Johnson [182]) was one of the first to state this relation clearly, since he was one of the first to show that a wound of the lung is by nature valvular, hence in many cases the expiratory sound can not be due to air leaving the chest. Such a case must certainly have been Variot and Roy's,[335] for the wound must soon have closed.

And again in some cases there was no question of the absence of a fistula. Powell (Johnson [182]) heard most typical amphoric respiration over a distended stomach, and Hoover [313] over a distended loop of intestine.

In two cases of Maréchal this was a marked feature, yet at autopsy the fistulæ were not found.

Cole [147] considered that in his case the point of maximum amphoric respiration and the fistula were identical. West [173] thinks his diagnosis of open pneumothorax based on very loud amphoric respiration confirmed in one case at autopsy. The same writer,[197] in another case, found loud amphoric breath sounds, yet a closed fistula. The sounds gradually disappeared with recovery. In Finney's [301] case it disappeared on the 5th day. Harris [205] and Finlay [320] considered in their cases the murmur due to an open fistula.

But that these sounds could be sometimes, if not always, produced at an open fistula many held. Laennec had heard them produced by a fistula opening externally. Stokes [70] had heard these same sounds. Weil [172] said if the sound were loud and like those produced by an external fistula it was evidence of a fistula opening internally. Johnson [182] said that amphoric and fistulous respiration due to an external fistula, might be of the same quality. Coupland [191] considered his case to present "fistulous respiration."

That such respiratory sounds could be present over a tuberculous cavity, granting it to be a large one (the fist size at least, Skoda), there was little dispute, although Barth (Hérard [100]) suggested in a discussion that such cavernous respiration was not amphoric.

Hence we may conclude, as a result of this short review of literature, that the question is not, "can this loud amphoric respiration be produced independently on an open fistula," but "can it ever depend on an open fistula?"

In the case of faint yet amphoric respiration the question is simpler. In this case the question of an open fistula need hardly be considered, although it may be just as truly open. It is hard to say how many of the writers just quoted really heard amphoric but faint sounds, sounds which Maillart and Lasserre [357] described as distant, amphoric, like the tones of a bell echoing along the arches of a cathedral.

Saussier [78] describes three classes of breath sounds, vesiculo-amphoric, broncho-amphoric, and fistulo-amphoric. The latter was the same as the amphoric respiration just considered, but the former two were sounds produced in the collapsed or compressed lung and intensified by the pneumothorax cavity.

Marais [90] took exception to Saussier's views, and thinks such sounds could have been produced only granting adhesions were present, since the partially collapsed lung was almost incapable of transmitting vibrations.

Piorry[54] (quoting Cayol) insisted that the breath sounds transmitted from the good side could be heard.

Johnson[182] reported a case with faint but distinct amphoric respiration with an occasional metallic tinkle, both of which disappeared thirteen days later as the case improved and the respiratory murmur reappeared.

And yet it should never be forgotten that entire absence of breath sounds has since the time of Laennec been considered one of the most important diagnostic points, especially of early pneumothorax.

In discussing the cause of these phenomena, it should be borne in mind that amphoric is the quality of the breath sounds and bears no relation to the intensity. The faintest murmur audible may be beautifully amphoric, or the breath sounds may be amphoric but loud and blowing.

That the breath sounds may acquire such quality, many factors may operate.

1. The original quality of the sound, and especially its pitch.

2. The presence of a resonance chamber which gives the sound its amphoric quality, and the physical properties of this cavity.

3. The nature of the medium between the source of sound and the resonance chamber.

4. The nature of the medium between the resonance chamber and the ear.

Loud Blowing Amphoric Breath Sounds.—These sounds are usually considered as occurring only in pneumothorax, and to be one of its most important signs. The great contention has been, is this sound produced by the air entering and leaving the pneumothorax cavity, or is this cavity merely a resonance chamber intensifying a sound produced in a neighboring cavity?

Such respiration was present in 10 of our cases (we include for the present all cases with loud breath sounds whether or not they were amphoric.

Case 1. Eight days before death inspiration was rough and harsh but not amphoric, and at the end of inspiration were present, distinctly metallic tinklings. Expiration was louder, rougher but distinctly amphoric. These amphoric sounds were heard in front as low as the seventh rib.

At this time the signs of distension were marked; the side was fuller,

almost immobile, the heart was much dislocated, (this was a case of right-sided disease) the liver was much depressed.

At autopsy the pleura was opened by a canula, but no intrapleural pressure then existed. The pleura was much thickened, and contained only about 500 cc. of pus. (The chest had not been aspirated).

The lung was completely collapsed, and compressed against the anterior mediastinum and the spine. Its tissue was almost wholly airless, almost the whole lobe in fact was either a solid caseous mass or a cavity.

The perforation was a large open one 10 x 7 mm. in diameter, and opened into a cavity which was an offshoot of one the size of a small orange.

This case may be summarized into these words: large resonance cavity, with thickened walls; large open perforation into a large pulmonary cavity; solid (compressed and consolidated) lung.

Case 3. Nine days before operation, over the whole front (recumbent) is intense tubular breathing; behind (erect) amphoric respiration extends to the angle of the scapula. At the base is fluid and absence of the breath sounds.

At the operation about one gallon of pus was evacuated.

At autopsy, the pleura was much thickened, the lung was airless and consolidated; the perforation was large, about 1 cm. in diameter, and opened into a large cavity.

Case 4. The auscultatory signs in this unexpected case were "cavernous" respiration to the 5th i. s. in the axilla and almost to the angle of the scapula behind; intense whispered voice and numerous liquid râles. The cavity must have been the pleura, for the lung occupied a small space, was compressed and consolidated. The perforation, size not stated, opened into a series of cavities; the pleura was much thickened.

Case 5. Three months before death were heard everywhere the most typical amphoric breathing with metallic tinkling down the back.

About two months before death, the sounds on coughing were amphoric but not so the breath sounds in front, but markedly so behind. (This continued much the same till death.)

One week before death, 600 cc. of pus were aspirated.

An autopsy were found much pus and little air, the pleura was very much thickened, the pyopneumothorax cavity very large. The lung was compressed to a small mass at the superior posterior part of the pleural cavity, and was both compressed and consolidated. Almost

the whole of the upper lobe was a cavity and the perforation into the pleura was 1 cm. in diameter.

In case 12 the breath sounds were amphoric but no autopsy was obtained.

Case 17. Here, an unrecognized case of circumscribed pneumothorax, the breath sounds over the area found to be the pneumothorax cavity were more intensely cavernous than over the rest of the lung. At autopsy this cavity contained 425 cc. of pus, and air. The perforation, 3 mm. in diameter, opened into a cavity the size of a walnut. The lung was riddled with cavities and the remaining tissue consolidated.

Case 25. Here "respiration was harsh, expiration prolonged." The lung was compressed above, consolidated below, with several perforations. The pleura was much thickened.

Case 26. Here intense amphoric breathing was heard only below the angle of the scapula, definitely amphoric below the 6th rib. At autopsy the lung was found small, compressed, airless. The perforation was at the base and 4 mm. in diameter. It was round with sharp edges, hence could not have acted as a valve.

Case 20. The signs here were interesting. There was no autopsy. In the upper part of the chest behind, the sounds were loud and cavernous above, and at operation the perforation was easily seen, for the operator drew a picture of the lung indicating the position in which he found it, hence it must have been both large and open.

Case 21. In this case the breath sounds were very loud and amphoric, the râles had a distinct metallic ring, and on coughing the râles had a characteristic amphoric quality. The patient recovered, hence had an open perforation existed it was soon closed.

Case 22. The signs are here marked. Here was loud amphoric respiration over the right front shortly before death. Previously they were distant and suppressed but still amphoric.

At autopsy, the pleural cavity was found a sac with thick, board-like walls. The lungs were greatly compressed; no perforation found.

Case 38. The breath sounds were described, as intense but still retaining their tubular modification. At the angle of the scapula they were hollow and amphoric. Amphoric but distant breath sounds. In children the chest signs are very irregular.

Faint Amphoric Breath Sounds.

Case 6. The breath sounds were feeble, nowhere characteristically amphoric but in front were heard the most beautiful metallic tinklings.

The pleura was much thickened, the lung compressed and airless; a small perforation into a small cavity was present.

Case 7. The breath sounds at the apex were feeble, below the clavicle feeble with distant metallic râles, but over the whole front and axilla were amphoric on forced inspiration and especially on coughing. The voice sounds had a metallic ring.

The pleura was much thickened. The lung, much compressed, was consolidated. A small perforation was found by a probe to pass into a small cavity; no other cavity was mentioned. Such a perforation was probably valvular.

Case 10. The breath sounds in this case were extremely feeble or not heard at all. Along the sternal border can be heard the faintest amphoric sounds. This was three hours before death.

The lung was only partly solidified; there was extensive cavity formation and into the largest, a very large cavity, a perforation 3 mm. in diameter. This case seems an exception to the rule.

Case 11. Here the breath sounds are everywhere very feeble, accompanied by râles piping and musical but not metallic, but although somewhat tubular in the axilla are nowhere truly amphoric.

The pleura was thickened, the lung, compressed and airless was riddled with cavities some almond size into one of which opened a large perforation 1 cm. in diameter into the pleura.

Cace 13. The breath sounds are almost entirely suppressed, but when heard have an amphoric quality. The voice sounds are weak with a slight amphoric quality.

The lung was much contracted; no fluid was in the pleural cavity, there was no evidence of cavity formation, the fistula was not found.

Case 14. The breath sounds were everywhere feeble; inspiration was slightly high-pitched, but expiration had a most marked bell-like amphoric quality.

The pleural cavity was very large; but 500 cc. of pus was present (none had been aspirated). The lung was small, fibrous, airless; a cavity the size of a hen's egg was present. The positive pressure found at autopsy by the escape of gas shows that the perforation which was present may have been valvular.

Case 12. The breath sounds were distant and suppressed. But little fluid was present in the pleural cavity. The pleura was slightly thickened, the lung compressed but not consolidated. The presence of a cavity to explain the sinus was supposed, at any rate the sinus must have been valvular.

Case 15. In this case the feature present was the faintness yet amphoric character of the breath sounds, and the beautiful metallic tinkling. The pleura was much thickened. The lung was compressed, airless, with cavity formation, yet the positive pressure twice demonstrated shows the fistula to be valvular.

Case 16. In this case the breath sounds over the base were distant but of a pure amphoric quality with metallic râles on inspiration, and sonorous on expiration.

The pleura was thick; the pneumothorax cavity was small, circumscribed, with thick walls. No perforation into the lung was found. The lung was extensively but not entirely consolidated.

Case 18. Here the signs of the pneumothorax cavity were overshadowed by those of a larger intrapulmonary cavity.

Case 40. The sounds were distinct, amphoric only on deep inspiration, not amphoric in the back.

Case 48. Is doubtful. If pneumothorax was present the fistula was not present at autopsy. The pleura was much thickened.

Cases 28, 42, 44, 46, all with recovery had distant breath sounds.

Among the pleurisy cases there can be little doubt that:

1. The fistula soon closed especially if made by the aspirating needle.

2. The lung was not perfectly airless, in fact probably took no small part in respiration.

3. If airless was not perfectly consolidated.

The following observations were made:

In cases 30, 31, 32, 35, 37, 39, all with recovery, the breath sounds were feeble, or absent. In cases 32, 37, the amphoric quality was also noted.

Case 33. The breath sounds were distant and obscure, the lung was aerated throughout. The air in this case entered from without and the lung not injured.

Case 34. The breath sounds were remarkably feeble, yet on deep inspiration had a distant amphoric quality. The lung was collapsed yet contained air throughout. No perforation was found, for the small needle prick must soon have healed.

Case 36. Here the breath sounds were everywhere remarkably feeble. The pleura was enormously thickened, the lung was extremely compressed yet could still float in water. No perforation was found.

Analysis of the above Cases.—These cases have been classified according to the intensity of the breath sounds, and secondarily, according to their amphoric quality. Believing the intensity the more important element, the least easily mistaken, for any sound may become amphoric if near a suitable resonance chamber, we shall continue that classification.

The condition of the pleural surface.—Of the twelve cases with intense breath sounds, the pleura was greatly thickened in six cases. In two cases of circumscribed pneumothorax the thickness of the pleura was not stated, but the presumption is that it was; in two cases there was no autopsy; two cases recovered. Hence in every case in which its condition was recorded it was thickened by fibrin and pus, or the organized exudate.

Of the twenty-six cases with recorded feeble breath sounds, in about thirteen it was thickened, counting two doubtful ones, in eleven cases the patient recovered. In two the condition was not stated.

Since cases 36 and 22 especially belong in this group, we fear the condition of the pleura will help us little, although this is doubtless one element in the result, but whether aiding or hindering is not certain. Bigelow,[76] however, gives evidence showing that the pathological pleura does aid in the production of these metallic phenomena.

The part played by the pneumothorax cavity.—In the production of amphoric respiration sounds, loud or faint, is a resonance cavity necessary? The answer is a positive no.

In the *first* place two elements could be supplied by a cavity. It is both a conducting and an intensifying chamber. Fluid is a much better conductor of sound than air, hence so far as the conduction is concerned the pneumothorax is a disadvantage. The amphoric quality in cases of pleural effusion is presumably supplied by the trachea and bronchial tree, and the fluid acts as a conductor. The following is a case in point. Especial attention is asked to this case since it is so similar to some reported as pneumothorax.

CASE 49.—*Tuberculous pleurisy; marked amphoric respiration; death; autopsy.*

Uriah B., colored, aged 44. **Med. No. 9486.**

Admitted January 23, 1899.

Died February 28, 1899.

Clinical Diagnosis.—*Tuberculous pleurisy; pulmonary thrombosis.*

The patient was admitted complaining of cough and pain in the right side.

His FAMILY HISTORY is negative so far as tuberculosis is concerned.

The PAST HISTORY is also negative, except his habits have generally been bad and he has been a heavy worker.

PRESENT ILLNESS began two weeks ago, at a time when he was feeling perfectly well. It began with cough and two days later pain in the right side on deep breathing. The cough was dry and hacking, the pain was severe. He has had no distinct chills, no expectoration at first, but recently a little, and on one occasion bloody.

PHYSICAL EXAMINATION.—The patient is a large-framed man, well nourished, no dyspnœa, no cyanosis. The thorax is well formed. There is distinct fulness throughout the right side of the chest reaching to the clavicle. The intecostal spaces are practically obliterated. Expansion on this side is diminished. On the right side vocal fremitus is felt as low as the third space, below which it diminishes and becomes almost absent in the lower axilla. The percussion note in the supraclavicular fossa is markedly impaired and has a suggestive Skodaic quality. In the recumbent posture the note in the first interspace is slightly resonant, but becomes flat when the patient is in the erect posture. Flatness is present throughout the whole of the right front and axilla. The right back is considerably more prominent than the left. Vocal fremitus is felt over the whole right back, but is relatively diminished. Percussion note is flat from apex to base. On auscultation the sounds have a distant tubular quality, and the voice sounds, especially below the spine of the scapula, are exaggerated and have a nasal quality. The left side is practically normal.

Heart.—Point of maximum impulse is in the 5th interspace 10 cm. from the midsternal line. The liver dulness does not extend below the costal margin. The abdomen is negative. The sputum is quite liquid and serous in character, some muco-purulent portions. It is not blood-tinged. Exploratory puncture was made at the right back in the 8th interspace. Clear serous fluid was removed. Leucocytes, 6600.

January 26. Dr. Futcher: The patient is more dyspnœic than two days ago. The temperature was elevated, ranging between 101 and 103. Expansion on the right side is markedly diminished. Vocal fremitus is felt over the upper right front, but diminished as compared with the opposite side. It is markedly diminished throughout the lower axillary region. Percussion note is impaired above the clavicle and practically flat throughout the whole front and axilla. On auscultation the breath sounds are quite well heard over the whole front and axilla. Respiration is high-pitched and has a peculiar amphoric quality. Vocal fremitus felt throughout right back, but everywhere diminished. Percussion note flat from apex to base. Respirations are everywhere heard and have a distant peculiar amphoric quality. Voice sounds are everywhere heard and have a peculiar character of ægophony except in the lower axillary region, where they are distinctly impaired. Point of maximum impulse is wavy and diffuse, most distinct 12½ cm. from the midsternal line. At 12.20 the patient was aspirated and 420 cc. of turbid fluid was obtained. No changes followed in the condition of the right lung.

January 28. Dr. Osler: There is flatness from apex to base on the right side, front and back. Vocal fremitus is felt over the entire right back. There is very intense amphoric breathing everywhere. There is no metallic tinkling, no trace of tympanitic quality even under the clavicle. The liver is not greatly depressed. The interspaces are a little obliterated. There is not complete immobility of the right side. The apex beat runs out like a shuttle. The coin sound is not obtained. No succussion splash obtained. The right side is 1 cm. larger than the left side.

February 4. Dr. Osler: The patient is a healthy-looking man. There is distinctly more movement on the left side but the right is not immobile. The apex beat is in the 5th interspace just outside the nipple line. There is no marked protrusion of the right side. There is communicated pulsation over the whole right mammillary region, not expansile, not palpable. The right chest is flat to the apex. Fremitus is well felt to the extreme base on the right back but is diminished. The sounds are not as intensely amphoric behind as formerly. Over the right front the amphoric character is most intense. The fremitus is much diminished.

February 11. Dr. Osler: The percussion note now has a distinct tympanitic quality. The apex beat is in the mid-line. The chest is flat as a board behind from apex to base, but fremitus is everywhere felt. Amphoric quality not so intense.

February 15. The sputum has been repeatedly examined but no tubercle bacilli have been found.

February 16. To decide the question whether this was a case of pneumothorax or not a needle connected with an aspirating apparatus which would collect the air was inserted into the right chest just at the angle of the scapula. Pressure before aspiration + 2 mm. of mercury; 700 cc. of fluid were aspirated. No air escaped. The pressure at the end was — 14 mm. of mercury. The fluid was hæmorrhagic.

February 25. Dr. Osler: There is hyperresonance over the right front. The breath sounds have lost their amphoric quality. Loud friction rub is present. The patient was repeatedly examined to determine if air were present or not. The succussion splash was never heard. The coin sound was never positive; metallic tinkling was never heard. There was flatness on percussion. The loud, intensely amphoric respiration and the persistence of vocal fremitus were considered as possible evidence of a case of pneumothorax in which the high tension of the gas produced the dulness on percussion. The respiration was so intensely amphoric that it was supposed that no condition except pneumothorax could possibly be present.

On February 28 the patient died suddenly with dyspnœa, extreme cyanosis. The temperature was constantly elevated, ranging from 100 to 103 until February 13 from which time it ranged from normal to 101.

The urine was negative.

Autopsy No. 1298.

Anatomical Diagnosis.—*Primary tuberculosis of pleura. Serofibrinohæmorrhagic pleurisy. Compression of right lung. Thrombosis of*

pulmonary artery. Congestion and œdema of lung. Tubercles in fascia of intercostal muscles. Splenic tumor. Slight perihepatitis.

Body 174 cm. long, strongly built, well nourished. Rigor-mortis present. Both sides of chest equal. Abdomen moderately distended.

Right pleural cavity opened with reference to the presence of air; none found; it contains 600 cc. of blood-stained fluid. There is fibrinous deposit on pleura, especially over lower segment of the thorax. On costal surface is a blood clot, 4 to 5 mm. in thickness, the size of palm of the hand. Visceral layer here thick with fibrinous adhesions.

The costal pleura on right side 4 to 5 mm. in thickness. The entire pleura is greatly thickened. It can be stripped off complete. On the thoracic cavity side are scattered miliary tubercles. On the costal wall side are conglomerate tubercles and congested vessels.

Tubercles are also present in fascia of intercostal muscles.

Right lung is compressed. Below it is bound to the diaphragm. Here thickened exudate 4 to 5 mm. in all lobes, which are united. There are even tubercles in adhesions. Extending into lung for 2 to 3 mm. is a nodule 2 x 2 cm. in extent, from the adhesions between lower and middle lobes. No other tubercles visible.

Left lung free and voluminous. Pleura is congested. On section much blood-stained serum escapes. Cut section much congested. In the main branch of the pulmonary artery is a mixed, recent blood-clot, which extends into secondary branches. Bronchi contains frothy blood-stained serum.

In this case the respiration was of a most marked amphoric quality. Bowditch (*Boston Med. & Surg. Jour.*, 1897, p. 386) reported four cases of pleurisy with intense tubular breathing and in one at least the term amphoric was used.

The *importance of a resonance cavity* is well seen in some cases in which the cavity was not the pleural, yet the breath sounds were amphoric; others in which sounds surely not produced in the pleura are much intensified by its resonating properties.

Second.—The pneumothorax cavity, if it is productive of loud, amphoric breath sounds, does so through its properties as a resonance chamber, and by harmonic vibration will intensify those sound waves of proper wave length and by its selective action in responding to certain overtones and not to others will change the quality of the sound. The result will be a tone first intensified, second modified, and this is called the amphoric respiration. In the cases reported the following may be noted, showing how closely the chest resembles a physical instrument.

1. The cases with distinct metallic tinkling were sometimes those with faint breath sounds.

2. The amphoric quality of the sound in case 1 was present on expiration, not on inspiration. This was also true in case 13, yet in the former the breath sounds were loud, in the latter faint. Case 14 is another excellent illustration; here inspiration was less intense than expiration with no amphoric quality, expiration prolonged and markedly amphoric.

3. The amphoric quality was sometimes present only on forced respiratory movements. Such was true of case 6, in which the sounds were feeble, and amphoric only on forced inspiration, and especially on coughing, and in which the voice sounds have a metallic ring; of case 21, in which the breath sounds were distant and amphoric only on deep respiration; of case 34, in which on deep inspiration distant amphoric breathing was heard; case 37, in which the amphoric quality was produced by coughing; in case 20 coughing had a metallic quality; and lastly case 22, in which an amphoric quality was present only at the base and on deep inspiration. In all of these cases the sounds were of course faint.

4. The amphoric sounds are not heard uniformly over the chest, unless very loud. In case 9 they are heard only along the sternal border. In case 10 the quality in the axilla was quite different from that in the front and back; in case 11 only in the upper front; case 18, there were present in the back only sounds of a pure amphoric nature. In case 21 the back is the very position where they were not amphoric. In case 26 they were intensely amphoric below the angle of the scapula; above, at the apex, was silence, and below the 6th rib they were amphoric but intensely so at one point; this was also true of case 38. In case 22, during the first attack, they were amphoric only at the base; during the second, down the front.

The Influence of Fluid Exudate.—Out of respect to Laennec this should be mentioned. The twelve cases with loud breath sounds showed large or relatively large effusions in eight cases, small in three, no effusion in one.

Of twenty-five cases with feeble breath sounds there was much or relatively much in eleven cases, a moderate amount (under 1000 cc.) in eight, none in six.

The Influence of an Open Fistula.—Since Laennec's discovery of the sign of amphoric respiration this has been one of the most disputed points. But a fistula is not necessary, for this breathing may be heard over another cavity than the chest, e. g. the stomach.

Study of our cases would indicate the following: In the first place, the rushing of air into and out of a cavity can be the cause of loud breath sounds. The loud tracheal murmur, the tubular respiration of a consolidated lung, are the common illustrations of this. In these cases, however, the size of the bronchial tree is usually not great enough to serve as a very good resonance chamber; yet in the case 49, cited above, it was amply sufficient. Again, in the case of the cavernous respiration of a vomica, how many would deny that that was due to the passage of air in and out of the bronchus communicating with this cavity; or that in the case of a large vomica the breathing may also be amphoric in some degree. In case therefore the air has free entrance and exit to a pleural cavity why may the most of the sound not be produced by that passage, as Laennec first taught, and the loud amphoric quality be contributed by the size and resonant properties of the cavity, granting that in other cases with just as loud amphoric respiration the resonance chamber be a closed one.

Those cases with loud breath sounds were the following, and it is interesting to note that here occur the majority of cases with large open fistulæ:

Case 1. In this the perforation was a large one, 10 x 7 mm. in diameter, and opened into a large vomica the size of a small orange. There was no sign of positive pressure in the chest at autopsy, hence we have here a large open fistula.

Case 3. Here the fistula was even larger, being 1 cm. in diameter, and opening into a large vomica.

Case 4. In this case the note says "a cavity communicated directly with the pleura."

Case 5. Here the whole upper lobe was found occupied by a cavity which communicated directly with pleura through a perforation 1 cm. in diameter.

Case 17. A perforation, 3 mm. in diameter, connected the circumscribed pneumothorax with a cavity the size of a walnut.

Case 27. The perforations into a solid gangrenous lung were present.

Case 25 had a perforation, round with sharp edges, 4 mm. in diameter, and perhaps the same could be said of case 20. It is unnecessary to add that in the case of a perforation into a cavity in a consolidated lung the valvular action of lung tissue can play no part. But in cases

21 and 22 there could have been no open perforation. We may perhaps be certain that in eight of eleven cases the open fistula may have furnished a part of the sounds which the cavity resonated.

In twenty-four cases the breath sounds were feeble. In sixteen of these the fistula was quite certainly closed; in six without much doubt valvular. In case 10 the perforation was 3 mm. in diameter and opened into a large cavity. In case 11 the perforation was 1 cm. in diameter and the water test showed no positive pressure, since the body must be shifted to cause the air to bubble out.

The next question to answer is, did any relation exist between the situation of the perforation and that in which the amphoric respiration was heard. In case 25 this was surely the case; in case 20 possibly so. In case 6 the relation is clear. In case 19 also. Yet in other cases there was localization of the sounds with amphoric quality which bore no relation to the position of the fistula, since this was closed, and there were other cases with open fistula and no localization of amphoric phenomena.

In conclusion, we may say that granting there must be a breath sound, and a resonance cavity to intensify this sound, there is no reason why the sound may not be produced at an open fistula, yet the perforation is no very important element.

The Influence of the Collapsed Lung.—That the condition of the lung of the affected side, the medium transmitting the sound to the pneumothorax cavity, was important, has long been recognized.

We may roughly divide the lungs into four groups:

1. The collapsed lungs, those still containing considerable air, which have assumed the size at which the elastic recoil of the tissue is just satisfied. Such a lung seldom exists, for it will, as a rule, be expanded somewhat with each inspiration and may play no small part in respiration.

2. The compressed lung, which is airless. The question of the fate of the air, whether pressed out or absorbed, will not trouble one who has tried with the hands to squeeze a small part of the lung until it is airless enough to sink in water; he will be sure that intrathoracic pressure never reduced the lung to that volume and degree of atelectasis. On the other hand, it is not at all certain that such lungs are merely airless; they are often described as "fibroid," "indurated." Yet in some other cases the lung, which perhaps was in this condi-

tion or at least was collapsed for a long time, will re-expand; the induration could not have been great.

3. Consolidated lungs. The processes usually present are tubercle mass formation, caseous or gelatinous pneumonia, perhaps other forms of consolidation.

4. Lungs with much cavity formation, for these must be physically a very different transmitting medium from a solid lung.

1. *Cases with Loud Breath Sounds.*—Cases 1, 3, 4, 5 will all answer well to one description; a small lung, airless, consolidated by the tuberculous process, extensive cavity formation.

Case 2 was not examined post-mortem, hence we do not know the condition of the lung except from the clinical symptoms and signs, and these speak for a very extensive process.

Case 17. The lung not "compressed," since the pleural condition was local, was nevertheless solid and riddled with cavities.

Case 27. The lung above was compressed and airless, below gangrenous and solid.

Case 25. The lung was compressed and sinks in water, hence airless.

Case 20. The lung at operation was found "entirely collapsed."

Case 22. The lung was "greatly compressed." Cases 38 and 21 recovered.

2. *Cases with Faint Breath Sounds.*—Here every condition is found. Of these twenty-five cases in nine the lungs were small, much consolidated and with cavity formation; cases 6, 7, 10, 11, 14, 12, 15, 16, and 18; in one case the lung was airless and consolidated, yet no cavity formation, case 13; in one the lung was compressed, with an abscess cavity present, case 48; one lung showed considerable broncho-pneumonia, case 42. In case 30 the lung was compressed; in cases 33, 34, 36 simply collapsed; while 40, 28, 44, 46, 32, 35, 37, 39, recovered. Of the remaining case 31, there was no autopsy.

Evidently, then, from the consideration of cases in groups little can be learned.

But, although conclusions may evidently not be drawn by considering these cases in groups, we would call attention to one group so far as I know not before much studied, for although pneumothorax has often been caused by exploratory puncture of the chest or by aspiration but scant mention reaches literature. Here are reported 10 cases, in all of whom we may assume that the lungs were fairly normal, and

in case 34, that the pleura was also. With the exception of case 38 every one of these cases showed feeble breath sounds. This one exception was a child 10 years old, and since in children the signs are so different from adults that they should be grouped separately, we propose to disregard this case. Five of these cases came to autopsy and showed collapsed, yet air-containing, normal lungs, and for the three who recovered so well, we will assume the same, and think case 31's trouble was chiefly in his pleura.

For these nine cases therefore we may claim normal collapsed lungs, not airless, and the absence of loud amphoric respiration in all, although the respiratory force was good, may be presented as part of the evidence that an air-containing normal lung is not suitable for its production.

To this group may be added case 28, in which the conditions of lung pleura and perforation were, if anything, even more normal than in the pleurisy cases.

But in some of these ten cases the feeble breath sounds were amphoric—cases 4, 6, 9, and 28.

The possible *sources* of the sounds which reach the ear as amphoric, loud, are:

1. Sounds produced in the trachea and the bronchi, both of the sound and collapsed lung. The larger bronchi in the latter will perhaps always be patent, and it is not necessary to assume that the sounds are always transmitted from the sound lung; on the other hand, Saussier's classification is very suggestive, as consideration of our cases due to aspiration will show.

The cavernous nature of these sounds produced in the bronchi has more than once led the clinician to diagnose a large cavity, while the pathologist discovers a solid lung. The pseudo-cavernous signs are too well recognized to need comment, and are due to the proximity of a consolidated area to a large bronchus.

The amphoric nature of these signs is not so often found, hence we have reported the above case as a marked illustration.

2. Cavities in the collapsed lung. Judging from the loud, cavernous, yet not amphoric respiration present in cases in which there is no pneumothorax but a vomica, an intrapulmonary cavity with a bronchus opening into it in a pneumothorax case should be a beautiful musical instrument to whose strains the resonant pneumothorax cavity should respond beautifully, adding the amphoric quality.

3. A condition very similar to this latter but with the pleural cavity instead of a vomica, into which the perforating bronchus opens.

4. All the above combined.

As a study of the literature and of these cases we would suggest the following CONCLUSIONS:

1. The factors which combine to influence the breath sounds are many; there are those which transmit the sound and those which modify it.

2. The loudness depends on the amount of effort exerted in respiration, on the good qualities of the intervening medium to transmit sound, and on the resonant qualities of the pneumothorax chamber to intensify it, or by intensifying certain components, modify it. The larger the cavity the more are the deeper harmonies and the fundamental tone intensified, hence the echoing reverberatory character upon which so much stress is laid.

3. The sound, however, produced, at the fistula, in a vomica, in the bronchi of the collapsed or of the sound lung, may be originally amphoric, as in some cases of pleural exudate; it may gain this amphoric quality by the harmonic vibration of a cavity, not the chest, e. g., the stomach; or the pneumothorax cavity may be the resonance chamber.

4. But if the pleural cavity does not respond well to these vibrations it will dampen the sound, for air is a much poorer conductor than fluid.

5. It may require vibrations of a certain amplitude to set the resonance chamber in vibration, hence the amphoric quality may be present only during expiration or forced respiratory movements.

6. A pneumothorax cavity bounded by a normal pleura is not so well adapted for this function as a pathological one, and the normal collapsed lung acts as a damper to the sounds. This conclusion we may draw from our series of cases due to aspiration.

7. For the production of such sounds the chest is a complicated musical instrument of which no one of the above discussed elements (fistula, etc.) is essential.

THE HEART SOUNDS.—The infrequency with which these sounds have a metallic quality has been explained (Leichtenstern) by their low pitch, hence for them the chest is not a suitable resonance chamber. They were metallic in but two of Weil's cases. In a remarkable case reported by Cornil [199] they were audible to bystanders.

In Morris' case [335] it was not the cardiac but exocardiac sounds of pneumopericarditis which were heard. Lundie reports a case of pneumothorax accompanied by pneumopericardium.

Frémissement Metallique. Strepitus Argentin.—This sign is produced by shaking the patient. The gas enclosed in the fluid escapes in little bubbles which burst on the surface and produce this sound. Strangely it is not described as related to metallic tinkling, although the conditions are fulfilled required by the old idea of a chest capable of giving the tinkling; that is, a submerged fistula allowing bubbles to escape through the fluid.

Coin Sound; Bruit d'Airain; Bell Sound.—This sign was first described in an anonymous article, the writer of which states that Trousseau introduced this method of examination. Trousseau [117] is said to have originally used a plessimeter and hammer on the front of the chest while the examiner put his ear against the back. Other means of percussion were mentioned, including the two coins which are now generally used. The sound produced was of such intensity that it "hurts the ear." He considered it pathognomic of pneumothorax. Skoda [116] had said that auscultation of the percussion tone was a valuable method in excluding emphysema.

A somewhat similar method of examination, the Plessimeter Stäbchenpercussion, Weil states was proposed by Leichtenstern. The thesis of Boursot [184] deals with the value of this sign. An early case in which the coin sound is mentioned is Gould's.[183] Harris [205] adds a note concerning it which seems to have failed of confirmation. Bruen [214] emphasized its value. Widal [216] stated that the sign varied with the tension of the gas, being absent when this was high and reappearing when diminished. It is a very early sign, being present at the very onset of pneumothorax before the metallic phenomena (e. g., Chauffard [274]), and its disappearance is one of the surest signs of recovery. Hoover [313] considered that it varied with the tension of the chest wall.

It should be borne in mind of course, that for the sound of the tap of the coins to be very clearly transmitted does not make the sign positive. The amphoric quality furnished by a resonant cavity is necessary. Béclère [295] said it became accentuated if the pressure increased.

Nevertheless the sign does not always mean pneumothorax, for Osler [255] has reported a case of tuberculous cavity occupying the upper lobe, in which it was beautifully heard.

Its presence or absence was noted in twenty-five of our cases. It was present in twenty, absent in three, suggestive in two. In case 35, due to aspiration of hydrothorax, a definite coin sound could not be definitely elicited six hours after the puncture. Three days later it was distinct, but eight days after the onset it had permanently disappeared. In case 37 the coin sound was not present at first but was very distinct four day later. In cases 31 and 38 it was only suggestive; in cases 10 and 22 it was not obtained; in case 16, a small circumscribed case, it was also absent. In case 15 it was so beautiful that it could be heard a short distance from the patient. In this case it could also be heard to about 10 cm. below the costal margin.

Metallic Tinkling.—Of all the signs of this disease this has perhaps been the subject of greatest dispute. The sounds are very characteristic; so beautiful are they that once heard they will later scarcely be mistaken.

Laennec described them as resembling the note made by striking with a pin or dropping sand into a vessel of glass, metal or porcelain; again as resembling the sound of the vibration of a metallic cord.

Andrew Duncan[52] described similar sounds but made no reference to Laennec in that connection.

The physicians in consultation with Johnson[58] said the sounds of a musical snuff-box gave a better comparison. Stokes[70] attributed to Graves the same description, but said a tuning-fork was a better one.

They were present, said Laennec,[41] during respiration, but also on speaking and especially well on coughing. They could, said he, occur simultaneously with or alternate with amphoric respiration. His idea that these two signs were due to the same cause, an open fistula into a fluid-containing cavity, was, he thought, confirmed by a case of external pneumothorax in which he could produce either at will by changing the size of the fistula. Scarcely ever was it present in hydropneumothorax without a fistula, except on change of position, in which case a sound was heard followed by the metallic tinkle and due to dropping of fluid from the surface of the thoracic cavity into the fluid below (case 40). He considered it more distinct, as the fistula was larger, and by the extent of the vibrations could the size of the air space be determined. It was present also in large tuberculous cavities. Laennec's description was excellent but his explanation has ever since been a favorite subject of discussion.

Williams [55] in his very lucid manner discusses these sounds at length. The falling drops theory he thinks may sometimes be acceptable, and saw one case in which he considered it sufficient, but pronounces such cases rare. According to him, the most important thing is a suitable resonance chamber with tense regular walls. An open fistula is an advantage, especially when short and narrow and the bronchus into which it enters large, but is not necessary. The sounds are usually produced outside the pleura, and are transmitted to the resonance cavity through the compressed lung. He [77] later denies that fluid need be present. It is audible in some cases in which no adhesions or fluid are present; in some in but one spot, in others everywhere. A certain degree of tension is necessary to make the wall a good reflecting surface.

Theories of the origin of metallic tinkling multiplied so that in 1839 Bigelow [76] was able to discuss those of Davies, Houghton, Guthrie, Bean, Spittal and Magendie. His conclusions may be stated as follows: Like Magendie, he could not produce these sounds post-mortem unless they had been present before death; an abnormal pleural cavity, one rendered suitable by a chronic process is necessary; in such a cavity the metallic tinkling is caused by the commotion of the fluid caused by coughing, etc.

Skoda's [81] explanation is the one usually given, yet this resembles in many ways Williams'; according to him metallic tinkling is merely the sound produced by the intensification of a râle by a resonance chamber.

Marais [90] reviews the subject, and accepts Skoda's explanation. He gives theoretical arguments why the fistula can have no part in the production of metallic tinkling, and why the falling drop theory is impracticable, but like all theoretical arguments these are merely interesting. He also discusses the opinions of Castelnau, Guérard, and Routier. The argument of Castelnau interests us, especially since this writer stated that if the lung be punctured air can be forced through the wound but cannot be aspirated in the opposite direction, hence, since the metallic tinkling is heard during expiration as well as during inspiration, the fistula, usually valvular in nature, cannot take part in its production. According to Marais the cause is a râle in the tracheo-bronchial tree to which the pleural cavity bears the same relation that a tuning fork does to its resonance box, and the metallic tinkle is comparable to an overtone, similar to that produced by a

sharp blow on a tuning fork and to which the resonance cavity responds.

He states that the sound produced by a submerged fistula is not metallic tinkling,

Milcent [97] defends the above views and discusses the opinions of Dance, Bean and Fournet.

Monneret [104] defends Laennec. According to him, the only difference between the tinkling and the succussion splash is in the intensity of the sound. Fluid is necessary, but a fistula not. He denies that the chest wall takes any part in their production, for if it did the hand pressed against the chest would dampen the sound, which is not the case.

In the decades following, now and then, one would find a case in which the "falling drops" theory seemed most satisfactory. Williams has been mentioned; Stokes [70] also saw such a case, and Liebermeister [230] states that Leichtenstern, by ante-mortem and post-mortem experiments was able to prove this origin in one case.

Rumpf [96] repeated that the tinkling was produced by the bursting of bubbles at the mouth of the fistula or at the surface of the fluid.

Others brought forth cases to show that no fistula was necessary. Forbes [64] states that in his best case of metallic tinkling it could not be found.

Others showed that no fluid need be present, e. g., Finney,[301] Maillart and Lasserre.[357]

In some cases it was present early in the attack, as in the case of Monneret,[104] Ross [168]; in some late, as in Louis' [48] cases 38 and 41, and Flint's.[115]

In some it was never present, as in Saussier's [84] case of pneumothorax due to a run-over accident, Church's,[151] M'Dowel's,[114] which was due to laughing, West's,[178] Adams',[202] Jaccond's,[204] and Chauffard's,[274] an almost spontaneous case. It will be noted that nearly all of these cases are of sudden pneumothorax in practically healthy men, due to injury or violence.

A few very interesting cases are those in which the tinkling was audible to the patient, although only through the stethoscope to the doctor. Such was Stokes' [70] case, and Fairfax's [137] patient was alarmed by the sounds.

But the most remarkable case of all was that mentioned by Allbutt (Finlay [320]) since in that the tinkling could be heard for two hours

in all parts of a large room. The patient, a healthy athlete, could hardly have had any previous effusion in the chest.

Among other interesting cases are Johnson's [105] in which from the second day the tinkling had been a feature of the case, and yet as soon as air was allowed to escape these beautiful sounds stopped. In Park's [119] case the tinkling was especially loud and at autopsy the fistula was found to open above the effusion. Bigelow replied that in nine-tenths of cases the tinkling was due to bubbles rising through the fluid and bursting at the surface, a long-held theory of the origin.

Unterricht [106] proposed a sign for the recognition of an open fistula opening below the level of the fluid, the Wasserpfeifengeräusch, produced by aspiration, the air bubbling through the fluid to replace the removed air and producing coarse, moist gurgling râles with a metallic ring. Riegel [169] heard a similar sound during the development of a pneumothorax. In these two cases is produced the condition which Bean, Bigelow and others considered sufficient for the production of metallic tinkling, yet neither described them as at all like this much-discussed sign; Bigelow might answer that the pleura was not enough altered, and yet Riegel described it in his case as much altered.

And, finally, metallic tinkling is present in other conditions. Hérard [100] reports it present in a tuberculous cavity larger than the fist, (also Laennec, Louis and others) and also [99] in a renal cyst containing both fluid and air. Biermer considered it of most value when heard in other than the interscapular and infraclavicular regions.

In our 39 cases in which the presence or absence of metallic tinkling was noted, it was noted present in twelve cases, of which seven were tuberculous cases, one a pulmonary abscess and three were cases of pneumothorax associated with pleurisy. Case 22 presented it during both his attacks.

It is of interest that the disease causing the pneumothorax was in every case of long duration, with the one exception of the abscess case.

Of the seven tuberculous cases, the average duration of the disease (tuberculosis not pneumothorax) before the date of the note describing the occurrence (we do not know always how long the tinkling had been present) was at lowest computation, almost two years, and of the briefest, seven months. Of the eight tuberculous cases in which it was not recorded, the average duration of the tuberculosis was about five months, and the longest case seven months. It was present in neither

of the cases of pneumothorax tuberculosa acuta, nor in the spontaneous case, nor in any of the cases following aspiration. In the latter cases the pleural cavity was certainly in many cases pathological, but the lungs normal. In the tuberculosis cases the reverse was more nearly true.

The cases were studied with reference to the extent of the disease, the presence of cavities, and the condition of the perforation, and but little was learned except that in the majority of the cases with fistulæ apparently open and not valvular, metallic tinkling was absent, four of the six cases with very large fistulæ coming in this group.

What was the relation of the tinkling to the amphoric respiration? Williams [55] considered that the latter would destroy the former. In seven cases these were feeble, in five loud. In case 6 they were beautiful in front, the sounds were feeble and nowhere amphoric. In case 7 they were heard distantly below the clavicle, the breath sounds were amphoric only on deep inspiration. In case 1 they were heard only on inspiration, the breath sounds were amphoric only on expiration; in case 5 they were heard only in the back; in case 15 they were beautiful over the lower front on coughing where the breath sounds were not heard at all; in case 16 they were heard on inspiration over the front while the breathing was pure amphoric only in the back; in case 25 they were heard over the lower front, the breathing was intensely amphoric below the spine of the scapula. All the above were cases due to pulmonary disease. In the three other cases all due to pleurisy (cases 32, 21, 22) they were evidently heard in the same area where the breathing was amphoric. In general then our tuberculous cases bear out well the opinion of Williams that there is a certain antagonism between this sign and amphoric respiration, also that the sign may be better heard in one situation than another. Williams thought it was best heard where the lungs were not adherent, and where the fluid did not come into contact with the chest wall.

In CONCLUSION we would describe metallic tinkling as produced by any short, high-pitched sound in the vicinity of a suitable resonance cavity, i. e., the pneumothorax cavity. The sound may be a râle, and perhaps usually is, since the occurrence of tinkling is the same as of râles, and it may be produced near or in the pleural cavity. There is no reason against and much in favor of the opinion that falling drops of fluid in the pleura, or indeed any sound, provided it be of the proper pitch and intensity, may cause the tinkling. The perforation need

not be open, fluid need not be present. Although the remarkable case described by Allbutt is an exception, we may agree with Bigelow, and our cases bear out his view perfectly, that the best resonance chamber is a pleural cavity the seat of long-standing disease.

The Succussion Splash is one of the most valuable signs for the presence of fluid combined with air in the pleural cavity. The class of pure pneumothorax has now reached such importance that the splash cannot be considered a necessary sign of the disease. Also the presence of a small amount of fluid may not give the splash, as Morgagni pointed out without knowing just whereof he spoke. Yet it is the earliest sign of fluid, and is heard distinctly when by no other sign, dulness for instance, can the presence of fluid be determined. This famous Hippocratic succussion splash, referred to so often and described as a noise like that in a leather bottle, in the complete works of Hippocrates although not in the genuine works, since to Hippocrates is denied the authorship of the chapters in which it is mentioned, was often heard and recorded during the two thousand or more years following, but its relation to pneumothorax only determined by Laennec. Since then it has become one of the most valuable signs of the coexistence of fluid with air in the pleura, and since these so often exist, of pneumothorax.

The intensity of the splash varies much in different cases. The writer of the chapters in Hippocrates' works must have met some very marked cases of the splash, judging from his description, since there it could be felt as well as heard. It was these marked cases, audible to the patient and to bystanders, which attracted attention until the time of Laennec, but they were regarded more as curiosities. It would seem that certain changes in the chest wall are necessary or else that the patient learns well the trick of producing a loud splash, for it is seen chiefly in cases of long standing duration, and in recent cases even though much fluid be present it requires careful auscultation, mediate or immediate, that it may be heard at all.

The noise that one of these long standing cases can make is remarkable. Houghton's [90] and Daland's [208a] cases could be heard to splash as they walked down stairs.

A few illustrations of such cases are Duncan's [52] case 3, Garde's [53] case 2. Banks' [120] was audible across the room, Davy's [44] also.

Morgagni discussed this sign at length and with considerable feeling. It would alone be enough, said he, to diagnose empyema

if always present, which unfortunately it is not, since just the right amount of fluid is seldom in the pleural cavity. Some, he stated, denied it ever occurred. He did not consider one justified in trying it. The sign really deserves Laennec's name. He showed its true meaning. According to him it is not difficult to obtain, shaking is not necessary, only a quick motion with sudden stop.

Laennec[41] considered its clearness depended on the amount of fluid present; if little the stethoscope was necessary, if more the unaided ear, if more still, it could be heard at a distance. Williams considered it best heard when there was much air and a moderate amount of fluid. Stokes[70] gives a most interesting hint, that in some cases it cannot be obtained in bed, but can be easily if the patient sit in a chair.

But the succussion splash may be present in conditions other than pneumothorax. Laennec cites one with a very large tuberculous cavity, and says the splash in the stomach may mislead. Wintrich had heard it for several weeks in a large tuberculous cavity.

It is very hard to compile any figures concerning the amount of fluid and the loudness of the succussion splash, since in very few cases would succussion be tried during the last few days of life on a patient who had shown a good splash formerly, and during these few days would the fluid in some cases increase rapidly. Hippocrates said those cases with less fluid gave a louder splash than those with much; Morgagni said some cases had too little, some too much. Neither knew the significance of the splash yet both were right, if we apply the remarks to pneumothorax. A most interesting case of our series is case 37. Here there was much fluid, little air. With the patient on his back the splash could be heard, but not when erect. It would have been interesting to have tried this in case 38, for with much fluid and little air the splash was not obtained. The customary way is to shake a patient when he is sitting up, and we may assume that was the method used in the case of that boy.

We have no case with too small an amount of fluid to give the splash. Case 7 may have been such a case since but 50 cc. of fluid were found at autopsy, but the last time the splash was noted absent was two days before death, and although the patient was perhaps shaken after then no note was made of it, and the small amount of fluid may have collected during that time. The absence in 35 is hard to explain. In cases 2 and 31 there was neither movable dulness nor the splash, but the amount of fluid is not known.

Cases 15 and 28 are particularly interesting, for in each the splash was beautifully heard before any other sign of fluid was evident.

In cases 31 and 22, almost our most chronic cases, the patients could themselves hear the splash.

Case 14 is one with an especially beautiful splash, yet the fluid could not have exceeded 500 cc.; in case 31 also, but here the fluid may have amounted nearly to two liters (three days later at autopsy 2200 cc. were found).

In case 32 the splash had disappeared in twelve days.

In case 28 it was noted for about the same length of time.

Gas Analysis.—That the gas in the pleural cavity was atmospheric air the surgeons had had little doubt, although the odor showed the anatomists especially that some at least was the result of decomposition. But Itard, Laennec and his followers believed in a gas of unknown composition and of disputed origin for their cases of idiopathic pneumothorax. It is only natural, therefore, that the chemist should interest himself in it.

Davy was the first. Fourteen hours after death he removed from the chest of one patient 212 cu. inches of gas, analysis of which gave CO_2 8 per cent, N 92 per cent. This observation led to animal experiments with the result that whatever gas or mixture of gas be introduced one soon finds a mixture of O, CO_2 and N. This led to still further [43] work to prove that there is normally none of these gases in the pleural cavity, and that the source of the N is in the blood. In another case Davy analyzed the gas four times removed during life and again after death.

Other early analyses were made by Apjohn. (Houghton.[65] See also Saussier.[78])

The work of Leconte and Demarquay [123] is particularly interesting and the first good series. Their method of determining the patency of the fistula by the increasing percentage of O in successively removed portions cannot be impugned.

Ewald [152] analyzed the gas of fifteen cases and determined that where the exudate is purulent the CO_2 per cent is higher than when serous. He describes an easy method for clinical use, and [153] reports a case in which it proved valuable. A little of his work was, however, done at the autopsy table. His average N per cent (79.81) is interesting.

Hoppe-Seyler [82a] did some very accurate and satisfactory work, to

the abstract of which the reader is referred. He emphasized the importance of the nature of the effusion and gave brilliant illustrations.

For other analyses see v. Ziemssen [189] and Ewart.[223]

But other gases than O, CO_2 and N may be present. Portal (Saussier [78]) allowed air to enter the chest at an empyema operation and soon found an inflammable gas containing H_2S. Duncan [52] was one of the first to determine chemically the presence of this latter gas, although the odor was sufficient for his predecessors and most of the followers. The presence of hydrogen was first settled by Hoppe-Seyler.[328]

Methods Employed.—The earlier writers used lime water and phosphorus as the dissolving bodies; the later the Hempel tubes with caustic potash and pyrogallic acid. Margerie [108] gave a good method of collecting gas. Ewald [152] an apparently easy clinical method for its determination. We have tried neither.

Concerning the gas which hisses from the chest at autopsy Marais makes an important suggestion. The air on entering the chest is warmed, hence expands; after death cools, hence contracts. If, therefore, gas hisses out, it must, some at least, have been formed post-mortem, and the high per cent of CO_2 found in such determinations bears out this point.

The opinion then seemed to be that the gas analysis furnishes some data in deciding whether the fistula is open or closed. Some attention had been paid to the character of the liquid exudate, and the gas has been analyzed whether it was removed from the patient before or after death.

In two of our cases the gas was analyzed. Case 13 showed 5.65 per cent CO_2, hence the perforation should have been valvular, and valvular it evidently was. In case 15 the analysis gave

CO_2.....13.5 per cent,
O.....14.4 per cent,
N.....72.1 per cent.

This should have been closed but valvular it surely was.

Six hours after death gas was removed for analysis. We found

CO_2.....30.9 per cent,
O..... 5.2 per cent,
N.....63.9 per cent.

We have investigated this matter at length in dogs. The gas analyzed was removed over mercury in Hempel's tubes and came in contact with but mercury until analyzed. The CO_2 was absorbed by NaOH, the O by pyrogallic acid. The directions in Hempel's book on gas analysis were exactly followed with the exception that mercury was used in the simple burettes. This requires much practice since the difference in level of the mercury in the two burettes should be carefully kept about the same. In the technic one thing should be emphasized; the scrupulous care necessary to have tubes well washed after each analysis that not a trace of $NaOH_2$ may cling to the glass to ruin the next determination.

1. Does the character of the fluid influence the nature of the gas? Ewald found it did.

Case 13 had no fluid; case 15 had much purulent. Some of the pus from this case was removed at autopsy and put into a flask suitably arranged that gas could be removed. The bottle contained 400 cc. of air, 150 cc. of pus. It was kept in the thermostat at blood temperature.

After 2½ hours,

CO_2..... 6.9 per cent,
O.....17.6 per cent,
N.....75.5 per cent.

After 24 hours (room temperature),

CO_2..... 9.4 per cent,
O.....14.7 per cent,
N.....75.9 per cent.

It is evident, therefore, that the presence of pus has a potent influence on the composition of the gas with which it comes in contact.

It will be noted that in case 15 during life the $CO_2 = 13.5$ per cent, and six hours after death and just twenty-four hours after the gas of the former analysis was removed, $CO_2 = 30.9$ per cent. There are evidently post-mortem changes in the gas. Is this true of dogs with pure pneumothorax? It certainly is.

Dog J. An open pneumothorax was first produced by inserting a cannula into the chest wall. After a few respirations this was closed for one hour. At that time,

$CO_2 = 7$ per cent,
$O = 15$ per cent,
$N = 78$ per cent.

The dog was at once killed, in 4 hours more gas was removed,

$CO_2 = 18.5$ per cent,
$O = 9.9$ per cent,
$N = 71.6$ per cent.

Dog G. A very large dog used for blood pressure experiments. Just after respirations stopped the cannula was inserted and the right lung allowed to collapse, aided by some artificial respiration.

After 18 hours,

$CO_2 = 22.2$ per cent,
$O = 4.5$ per cent,
$N = 73.3$ per cent.

This change occurred wholly after death.

Dog F. At death,

$CO_2 = 7.8$ per cent,
$O = 14.7$ per cent,
$N = 77.5$ per cent.

After 24 hours,

$CO_2 = 39$ per cent.
$O = 9$ per cent,
$N = 60.1$ per cent.

Dog M. Air allowed to enter while dying. In 19 hours,

$CO_2 = 11.4$ per cent,
$O = 9.5$ per cent,
$N = 79.1$ per cent.

Dog H. Just before death,

$CO_2 = 9$ per cent.
$O = 14.4$ per cent,
$N = 76.6$ per cent.

Three days later (cold room),

$CO_2 = 12.7$ per cent,
$O = 10.9$ per cent,
$N = 76.4$ per cent.

Hence in some cases the changes in composition of the gas after death are considerable. The cause is evident. There is a post-mortem production of CO_2 and that of the tissue bounding the pleural cavity is liberated into this cavity.

That during life the lung tissue is not merely a diffusion membrane but an organ which actively produces CO_2, see Bohr, *Arch. de Phys.*, 1897, pp. 590, 819.

It has been shown that if air or another gas be introduced into the pleura it rapidly comes to a quite uniform composition. To study this problem dogs were used. In several of these a further point was studied, namely, does the composition of the intrapleural gas depend directly upon that of the intrapulmonary air. If so, to suffocate the dog should change the composition of the intrapleural gas.

Dog D. Jan. 8, 1903. Air allowed to enter, cannula closed 2 min.,

$CO_2 = 1.9$ per cent,
$O = 18.8$ per cent,
$N = 79.3$ per cent.

The cannula was left open for 45 min. and then closed for 10 min.,

$CO_2 = 3.6$ per cent,
$O = 20$ per cent,
$N = 76.4$ per cent.

Dog E. Jan. 15, 1903. Air in chest 10 min.,

$CO_2 = 4.6$ per cent,
$O = 18.6$ per cent,
$N = 76.8$ per cent.

The trachea was now compressed 3 min., rendering respiration very difficult.

$CO_2 = 5.5$ per cent,
$O = 18.3$ per cent,
$N = 76.2$ per cent.

Dog F. Jan. 16, 1903. Cannula closed 15 min. after lung collapsed by air.

$CO_2 = 5.6$ per cent,
$O = 17.7$ per cent,
$N = 76.7$ per cent.

Closed 60 min.,

$CO_2 = 7.8$ per cent,
$O = 14.7$ per cent,
$N = 77.5$ per cent.

Dog H. Jan. 23, 1903. Closed 30 min.,
$CO_2 = 10.3$ per cent,
$O = 15.5$ per cent,
$N = 74.2$ per cent.

Dog suffocated 3 min.,
$CO_2 = 10.3$ per cent,
$O = 14.6$ per cent,
$N = 75.1$ per cent.

Dog allowed to breathe easily 10 min.,
$CO_2 = 9$ per cent,
$O = 14.4$ per cent,
$N = 76.6$ per cent.

Dog K. Jan 31, 1903. Cannula closed 45 min.,
$CO_2 = 5.5$ per cent,
$O = 17.7$ per cent,
$N = 76.8$ per cent.

Suffocated 7 min.,
$CO_2 = 6.9$ per cent,
$O = 16.2$ per cent,
$N = 76.9$ per cent.

Breathed freely 8 min.,
$CO_2 = 6.9$ per cent,
$O = 16.6$ per cent,
$N = 76.5$ per cent.

Dog I. Jan. 26, 1903. Cannula closed 60 min.,
$CO_2 = 1.6$ per cent,
$O = 16.9$ per cent,
81.5 per cent.

Suffocated 5 min.,
$CO_2 = 3.8$ per cent,
$O = 15.5$ per cent,
$N = 80.7$ per cent.

Breathed freely 10 min.,
$CO_2 = 3.4$ per cent,
$O = 15.4$ per cent,
$N = 81.2$ per cent.

Dog J. Jan. 31, 1903. Closed 1 hour,
$CO_2 = 7$ per cent,
$O = 15$ per cent,
$N = 78$ per cent.

Dog N. Feb. 11, 1903. Air in chest 3½ hours,
$CO_2 = 6.8$ per cent,
$O = 11.7$ per cent,
$N = 81.5$ per cent.

Dog L. Feb. 5, 1903. Air in chest 6 hours,
$CO_2 = 3.1$ per cent,
$O = 12.6$ per cent,
$N = 84.3$ per cent.

It is seen that suffocation does change the percentage composition of the gas, but to a much less degree than would be the case if there were free osmosis between the alveolæ and pleural cavity. The changes in the gas after various periods may also be well studied from these experiments.

See also the most interesting experiments of Rodet and Nicolas [250 280], and the opinion of Bunge [204a] concerning the absorption of gas.

Dog B. June 12, 1899. 326.4 cc. (in this case all amounts are reduced to 0° and 760 mm. pressure) introduced into chest.

June 13,
$CO_2 = 7.6$ per cent,
$O = 10.03$ per cent,
$N = 82.37$ per cent.

June 14,
$CO_2 = 7.8$ per cent,
$O = 11.8$ per cent,
$N = 80.4$ per cent.

June 15,
$CO_2 = 5.05$ per cent,
$O = 9.7$ per cent,
$N = 85.25$ per cent.

June 16,
$CO_2 = 7.2$ per cent,
$O = 8.9$ per cent,
$N = 83.9$ per cent.

June 17,

$CO_2 = 6.8$ per cent,
$O = 9.05$ per cent,
$N = 85.15$ per cent.

Dog C. June 14, 1899. A mixture of gas containing 98 per cent of oxygen was introduced.

June 15,

$CO_2 = 5.04$ per cent,
$O = 9.7$ per cent,
$N = 85.26$ per cent.

June 16,

$CO_2 = 6.8$ per cent,
$O = 13.5$ per cent,
$N = 80.7$ per cent.

Conclusions.—If air (or the other gases tried) be introduced into the chest of dogs there is an almost immediate accumulation of CO_2 and diminution of O in the pleura.

The N per cent is remarkably constant until a point at which one may suppose absorption of the gas is well under way, when the N per cent rises about 8 per cent and then remains quite constant.

The composition of the mixture depends on the gases of the blood and also on the local respiration of the tissues. This is shown by the slow and comparatively slight changes resulting from our suffocation experiments as well as by the work of others mentioned above.

That the local tissue respiration is to be considered is shown by the rapid accumulation of CO_2 after death.

In the case of man the following points may be made:

Gas analysis has very little value in the diagnosis of the condition of the fistula, for:

1. There is a rapid accumulation of CO_2 in the pleura after death, which fact rules out the majority of analyses yet published.

2. The presence of a purulent exudate is an important element in determining the composition of the gas.

3. This post-mortem accumulation of CO_2 may explain the high tension of the gas which hisses from the chest on the autopsy table.

4. The method of diagnosing an open fistula proposed by Leconte and Demarquay [126] seems valid.

So far as we know these points have not before been made.

The Effect of Gas on the Pleura.—It was at first quite unanimously believed that air in the pleura was an inflammatory agent (Houghton [65]) although Laennec maintained that the "animalized" gas present in idiopathic cases was not. Physically the results would be almost the same as those of fluid (Benj. Bell [34]).

Davy demonstrated beyond question that for dogs air does not cause pleurisy. Stokes [70] was of this opinion; Wintrich [111] confirmed it experimentally. Proust,[124] however, held a contrary opinion and because of this error condemns as not pneumothorax some well reported cases.

Demarquay's [129] suggestion is very interesting, that a mixture of much CO_2 and little O is not irritating and is more easily absorbed than air; but if the air be constantly renewed so that it is in contact with the pleura for some time and maintains a high O per cent, it is an irritating agent. Widal [216] attributes this idea to Vieuille. Other gases are of course irritating, H_2S, $(NH_4)_2S$ and pure O.

Carbon dioxide, considered by Solon as a dangerous gas, is now considered as the bland element of the mixture and also as the antiseptic of the pleural cavity.

Demarquay [129] called attention to the acute exacerbation of symptoms following the formation of a pneumothorax in a tuberculous case, and Merklen [296] also mentions this point, giving an excellent temperature chart.

Nevertheless the pleural cavity often does remain dry, in spontaneous cases usually, in tuberculous cases often, even though the condition persist nine weeks (Weil,[180] Fussell and Riesman,[350] who found but one of fifty-eight cases with fluid). Adams [202] states that a case remained pure for four years.

Of course if for any reason the pleura is the seat of an empyema before the rupture of the lung or pyopneumothorax develops, there follow all the changes, thickening of the pleura, etc., so common in empyema. The lung so contracted would be expected to be rendered incapable of re-expansion, since the adhesions and thickening of its pleura would prevent its expansion. West considers this fear often more theoretical than real, although every series of reported cases contain some such, and in some cases the lung is so covered by false membrane that it cannot be found at first. Alexander [49] reported such a case and the editor of that journal explained his and mentioned another. Duncan's [82] and Garde's [83]

also were similar. Hence four such cases were noted in two years. Since then they were perhaps too common to report.

Nature of the Effusion.—Not only will the pleural cavity tolerate gas for a long time without a pleural effusion, but the effusion when once it is present may remain serous for a long time, and if purulent seldom becomes putrid.

Stokes [70] noted that the effusion might remain constant for months or increase steadily. In one case the fluid remained serous five months. Desplats,[168] finding one case remained serous after tapping, was interested to look up the matter and found seven cases in literature with such an effusion.

Weil,[180] Senator [242] said that there was no case of pneumothorax opening externally without a purulent exudate, but why the pleural cavity should be so resistant to infection through a wound opening through the lung was an interesting question. Auquier [175] attributed it to the lack of oxygen, rather a poor reason since the bacteria may be anærobic.

Augry [208] said the air was sterilized by the moist mucous membrane. Widal [216] accredits Lister, Strauss, Dubrenith with a similar explanation, the organisms being filtered out during the passage of the air through the bronchial tree. Senator [242] believed the lung to be a good filter. In children the effusion was more often purulent than in the adult. He also calls attention for the first time to the association of bilateral pleurisy with unilateral pneumothorax. Senator also believed in the antiseptic properties of CO_2. As a result of these two factors, said he, a large empyema cavity rupturing through the lung tissue seldom becomes putrid, while this is often the case if it ruptures directly into a bronchus, since the filtering action of the lung is less perfect and the constant renewal of air prevents a high percentage of CO_2.

West [287] found in his review of cases the fluid serous in one-third, seropurulent in one-third and purulent in the remaining third.

The cases of hæmopneumothorax are of especial interest. Pitts [333] has reviewed these at length. The most interesting one found is that of Boland [329] in which quite pure uncoagulated blood was twice aspirated.

A case of particular interest was Stokes',[70] who was able to evacuate the contents of the pleural cavity by almost standing on his head.

The Absorption of Air.—This for normal pleural cavities is considerable. Nysten (Duncan [52]) was evidently one of the first to emphasize this point.

Skoda [116] early showed that air was rapidly absorbed by rabbits.

Bunge [203a] has given an interesting explanation for the absorption of gas.

That the composition of gas during absorption remains fairly constant, dog B is advanced as evidence.

For inflamed pleuræ, by far the most interesting to us, it was studied by Szupak,[239] who found the rapidity varied inversely to the degree of inflammation, that pure O or pure CO_2 were absorbed in the same time as air, but pure N only one-half as fast.

Rodet and Nicolas [259] found absorption rapid at first, slower later, and complete in about one month (for dogs).

Unterricht [266] said a dog could absorb the most of the gas in twenty-four hours.

Examination with the Röntgen Rays.—The value of the photograph is well seen in the cases of Stembo,[317] Williams,[321] Martin,[340] and Jochmann,[351] and the progress of the case may be thus well followed.

Radioscopy seems especially valuable, even the waves on the surface of the fluid may be seen (Williams), and it was in this way that the paralysis of the muscles of respiration was recognized. (Maillart and Lasserre.[357])

CHAPTER V.

Course, Symptoms, Prognosis, Diagnosis, and Treatment.

Onset.—This may be very sudden and severe, or one may be able to date it by questioning the patient, or it may have developed without any symptom. Laennec mentions illustrations of each.

Louis [45] lays great stress on the onset of tuberculous cases, and states that from the history alone one may diagnose this condition in a tuberculous patient.

Saussier reports a sudden onset in 68 of the 196 cases he collected.

The general rule may be made that the more normal the lung the severer the symptoms of its rupture, hence in case of spontaneous pneumothorax or that due to the rupture of an emphysematous bleb

in an otherwise normal lung, the onset is extremely severe. Such cases were those of Church [151] whose patient was at the point of death; of Ross,[168] whose patient was unconscious for an hour, and of Klemperer [251] also.

Other cases while not unconscious, suffer extreme pain at the onset. Delafield's [318] patient felt as if his "spine were squeezed in a vise;" but the case which interests us especially was that of Maillart and Lasserre [357] whose patient held himself motionless for a while unable to stir, for this reminds us of one case of pneumothorax we saw. An eminent clinician on learning the man's story, evidently supposed at first from the description of the onset that he had to do with a case of angina pectoris and asked if the sudden pain ran down the arm to the ring and little finger, and remarked on the case as if he had that disease. On examining the chest, however, he found a clear case of spontaneous pneumothorax in a quite healthy man.

Some cases can feel the rupture; as four cases collected by Saussier. Stokes' [70] case is an illustration; his patient felt the crack and had the feeling of liquid shed into the chest; also Boland's,[329] and those of others. In other cases the rupture is audible; Banks [110] reported such a case and finds in literature three others.

In case, however, the lung was extensively diseased, its perforation and collapse to small volume may occasion no marked symptoms and an increasing dyspnœa be the only result, and even this may fail.

But even the typical onset may not denote pneumothorax, as Nothnagel [248] said, who mentions one case in which the rupture of the contents of a tuberculous cavity into a bronchus caused the same symptoms.

And dyspnœa is an even more important symptom of pneumothorax (Laennec [41]) than is pain. This was one of Itard's [80] symptom-complex. In some cases it rapidly reaches extreme grades (Gould [185] respirations 60 to the minute, Galliard [243] 64 to the minute).

This pain and dyspnœa have been attributed to the tear of the pleura (Saussier,[78] Gérard [127a]) and also to the pleurisy which will be at once excited if the contents of a cavity be poured into the pleura. Stokes [70] said it was not due to the exclusion of the lung since it was absent in cases of occlusion of a primary bronchus, but was of nervous origin due to spasm of the good lung and to pleurisy. Demarquay [129] insisted that in cases with emphysema it was due to the embarrassment of the muscles of respiration and not to the com-

pression of the lung as Hewson thought, since it is present in cases in which adhesions prevent collapse of the lung. Hewson's error, he said, was pointed out by Malgaigne.

Another symptom at onset was early pointed out by Stokes,[70] the change in the character of the cough and the cessation of sputum especially if the source of the latter was the perforating lung. He mentions one case in which a copious expectoration ceased, only to return as the other lung became involved. In this case it was diminished formation. In another case the sputum seemed to fall into the pleural cavity.

The Course of the case will depend chiefly on the disease of which the pneumothorax is a complication. So far as pneumothorax alone is concerned, soon after the onset the person, unless it be a case of suffocative pneumothorax, will develop in less than one week (Stokes[70]) a remarkable tolerance to the condition, (see Bouveret[211]). Laennec considers dyspnœa the most constant symptom but even that may be absent (Murdoch,[244] Thornburn,[120]). Fever is rarely present, (Fussell and Riesman[150]).

The onset, course and prognosis led several early writers to compare the perforation of the lung with that of the intestine (Houghton[60]), and the resemblance is in many cases striking.

How great the tolerance which soon develops may be is illustrated by Stokes'[70] case of a man with tuberculous pyopneumothorax who insisted he was well, yet complained of a splashing sound heard while dancing or riding horseback. Weil[181] reported such a case who lived $2\frac{3}{4}$ years after leaving the hospital, but this now seems a short time. The duration in cases of pyopneumothorax is of course the shortest, while it would seem that a pure case could continue for years.

The Prognosis of the case will of course depend much on the causative disease, but of the condition itself, it is good. The mortality will of course depend on the frequency with which cases are recognized. Laennec who had never seen a case not in bed could have recognized none of the well marked but latent cases with which the literature of the past two decades has become sprinkled, and yet Laennec believed that the prognosis was not hopeless. Houghton published a case which he considered unique, of a brick-layer able to work for some time, although with pyopneumothorax, and whose fistula showed some signs of repair. Poole[98] considered a case with such signs of repair

as worthy of publication. Stokes [71] considered the cures only apparent and the limit of life about one year.

Biermer [121] in 1860 stated that at that time the unimportance of pneumothorax per se was recognized, but that cures of non-traumatic cases were so rare that Lebert, who collected 6000 (!) cases, doubted the diagnosis of any reported recovery.

In 1883 Biach [190] collected all cases of recovery he could find and later West [197] made a collection of a certain variety of healed cases. The longest had a duration of eleven weeks. The conditions controlling the prognosis are cited by Bouffaré.[210]

Saussier [78] gave a table of the duration of 91 cases.

West [193] stated the general mortality as 65.4 per cent, of whom 75 per cent died in 2 weeks and 90 per cent in the first month. The same author [287] later gave the mortality as 70 per cent, discussed the various causes of death and reported eleven cases with recovery. He mentioned one case, the lung of which had been collapsed for eighteen months, yet re-expanded. West considered that the prognosis rests much with the other lung, and if râles are heard here the prognosis is bad.

Augry [205] found that three of nine cases due to emphysema died, the death being due either to double pneumothorax or the inability of the one emphysematous lung to keep up sufficiently the respiration.

The prognosis of suffocative cases, happily rare, is without operation bad. Such cases are said "to progress to death." That of the "spontaneous" cases excellent, Fussell and Riesman [350] report that all recover.

Such cases are those of Noble [122] with recovery in two months, Wilks,[148] Gibson [273] in two weeks, Weber [332] in 32 days, Johnson [162] in 7 weeks, and others.

Szupak [289] stated the time in such cases to be 26 to 60 days, and mentions Nonne's case, which was well in 16 days.

Traumatic cases also recover rapidly; that of Variot and Roy [335] in 23 days, West [257] in 16 days, Cayley [174] in 16 days, Butlin [150] in 5 weeks.

It is unnecessary to add that the duration of such cases depends chiefly on the judgment of the examiner. Some cases are dismissed when the symptoms have entirely disappeared, a much too short time; others when certain signs are no longer present, e. g., the coin sound, the amphoric phenomena, or when the heart has returned to its normal situation.

Nichols [224] considers the course more rapid in children than adults. Widal [216] considered that the fistula would close in 8 to 15 days.

The methods of termination were given by Da Costa [140] as recovery, the disappearance of air but the permanence of the pleurisy, or the case may become chronic and the person enjoy good health for months.

Diagnosis.—For this the symptoms alone are not sufficient (Stokes [70]), and that all the other physical signs except auscultation would not suffice, the history of the disease shows.

It is interesting to note that the point which Laennec emphasized most is one often overlooked in discussing the diagnosis of pneumothorax; the most important point is the resonant percussion note on a side where the breath sounds are distant or absent. Nothing said of metallic tinkling, amphoric respiration, etc. Those two points constitute his symptom-complex of pure pneumothorax. Weil takes this stand also, but specified that the hyperresonance must be beyond the limits of normal resonance.

If fluid exudate was also present, to the resonance on percussion and absence of breath sounds Laennec added movable dulness and the succussion splash.

If also the fistula was open, to these must be added metallic tinkling and amphoric respiration, "the positive and the only signs of this triple lesion."

Laennec considered that no other condition could be confounded with pure pneumothorax or closed pneumothorax with liquid exudate. But we may well question if the diagnosis is as easy as this. Laennec described twelve cases as types and often states that these were selected cases and he could have multiplied such illustrations, in one case by even thirty similar cases. One would imagine from his writings that he had seen nearly or quite one hundred cases and this in a period of very few years from the discovery of his stethoscope to the publication of his book. Judging from the number of cases which large tuberculosis clinics are able to report, this number to have been seen in so short a time by one man is scarcely credible. For one of his diagnoses (No. 36) Proust calls him strictly to account.

Following Laennec's publication, clinicians on all sides began to report cases. Louis re-reported some of his cases with a change of diagnosis. The condition was found due to many various causes. It is only to be expected that many of these were not cases of pneumothorax. It was the thesis of Proust that cautioned practitioners. One of the disease mistaken for pneumothorax was:

1. Pneumonia. Graves [66] reported a case of "pneumothorax due to pneumonia" with a clear, hollow sound just below the right nipple and without respiratory murmur. On the next day the note was dull and the breath sounds audible. Williams [77] pronounced this tone the "tracheal tone," due to consolidation of the upper lobe. Biermer and Wintrich [111] both ascribe to Williams the credit of first describing this note heard over a pneumonic consolidation or pleural effusion which usually bears Skoda's name since he first gave its correct explanation. Wintrich describes it as follows: In the center of or beside a dull area the note on percussion is so pure and metallic that it is easily mistaken for pneumothorax. Such a tone is always anterior and especially just below the clavicle or in the mammary region. But the fremitus persists and the râles and breath sounds are not metallic. At the apex can be obtained a beautiful metallic note, Williams' tracheal tone, but the change of pitch on opening the mouth will be present.

Biermer [121] said there was some doubt of the value of the amphoric phenomena since they were also present in pleurisy and pneumonia, as Williams first showed, but in these no metallic phenomena occurred, and the amphoric respiration would be clearest at the root and apex of the lung, would not be so loud or exquisite as in pneumothorax or a tuberculous cavity, and would diminish on quiet breathing.

2. Pleurisy with effusion. It may require the needle to settle the presence of gas in these cases, as in the one (49) cited. The Skodaic note of the collapsed lung may be beautifully amphoric. This was particularly so in the case of a child recently in this hospital; the note which Dr. Osler was able to produce was so amphoric that it was hard to believe that air was not present.

Strange as it may seem, it is not always an easy matter to determine experimentally the presence of pneumothorax in a patient. The little instrument of Béclère is excellent. If an aspirator is used one may be much deceived, as we have been, for under the negative pressure joints apparently tight will leak badly. We have used with good advantage an apparatus to collect the gas, which consisted of a flask (500 cc.) with a rubber stopper with two tubes, one passed to the exhaust bottle, the other was a three-way-valve and connected with the chest and a manometer. The flask was first allowed to fill with normal salt solution and when the last bubble of gas had left was inverted and suspended in a ring on a burette stand, a segment of which ring had

been cut out to allow the flask to be slipped in. The chest could then be connected with the manometer or with the flask, and when aspirating any bubbles of air will collect in the inverted flask.

3. A tuberculous cavity. Itard [30] gave the points in the differential diagnosis of these conditions. Laennec [41] admitted one mistake, on which occasion he based his diagnosis on the succussion splash. The lower two-thirds of the right lung was occupied by a great cavity. Louis[48] evidently made a correct diagnosis in a case with clear percussion note and distinct metallic tinkling, but also with pectoriloquy and tracheal respiration. In a similar case Wintrich could obtain the succussion splash for weeks, and admits to have mistaken cavity for pneumothorax, localized pneumothorax for cavity. Nothnagel considers the intensity of succussion important. Galliard [261] discusses the question more at length. The hyperresonance, succussion splash, amphoric respiration, metallic tinkling, coin sound, any of these, perhaps all at once, may be present in a large cavity. The points of difference are that in cavity the chest is not so immobile and not distended, the heart and liver are not displaced, the intercostal spaces not so obliterated, movable dulness to such a degree will not be present, the amphoric phenomena will not be present over so wide an area, there will be some remains of pectoriloquy, on coughing a succussion splash may be heard (never present in pneumothorax—Laennec), the cracked-pot sound and Wintrich change of pitch may of course be present in both, and usually the respiration will be not so beautifully amphoric. Galliard considers the position of the heart and liver of greatest importance.

Laennec [41] thought that to feel the succussion with the hand was to exclude a large cavity.

Nothnagel [248] cites one very interesting case in which the stomach furnished the succussion splash, and the rupture of a cavity into the bronchi the clinical symptoms of pneumothorax. He warns one to be suspicious of the signs of the latter at the apex.

4. Emphysema. Itard and Laennec both considered this diagnosis should be easy, yet Nothnagel [243] mentions a case with the signs of pneumothorax. It is the character of the breath sounds and the absence of metallic phenomena which should give the diagnosis. Skoda adds the auscultation of the percussion tone.

5. Diaphragmatic Hernia. Butlin's [150] case is of particular interest, the diagnosis was made only on the autopsy table.

6. Pneumothorax Subphrenicus. Of this we give one example:

John Sp., white, aged 36. **Med. No. 331.**

Admitted January 16, 1890.

Discharged January 27, 1890.

DIAGNOSIS.—*Pyopneumothorax subphrenicus.*

The patient was admitted COMPLAINING of fever, diarrhœa and pain in the abdomen.

The FAMILY HISTORY is negative.

The PAST HISTORY is negative. He has never had a previous illness, save typhoid fever when 12 years old. He has been a hard drinker.

PRESENT ILLNESS.—Last September dysentery began to trouble him somewhat. There has been much blood and mucus at times and some pain. The stools have varied from two to twelve in number daily. There has been some vomiting but not marked. The pain in the abdomen is constant. It is in the hepatic region and extends from the epigastrium to the back. There is no swelling in this region. He has lost 60 pounds in weight and much strength. He is short of breath. His legs are œdematous at times. Thinks he has had fever for six weeks, but has worked off and on. Has had no chills. He has had cough for but a few days; has never been jaundiced; never had a severe sharp pain in the region of the liver.

PHYSICAL EXAMINATION.—The patient is emaciated and anæmic. Respirations 30; pulse 96. The chest expansion is good; equal above but greater at the left base.

Percussion.—The anterior is normal. Posteriorly there is slight dulness at the extreme right apex, perhaps a slight increase of tactile fremitus.

Auscultation.—The breath sounds are a little feeble but no râles are heard at the right apex. At the right base a few dry crepitations on deep inspiration, probably pleuritic.

Liver dulness begins at the 5th rib and extends to 1¼ inches below the costal margin. The edge cannot be distinctly made out. The surface is not rough or tender. In the median line of the abdomen the liver dulness extends to 1¼ inches below the tip of the ensiform. The spleen is not palpable. The abdomen is a little full and tympanitic but not tender. It is symmetrical.

Urine shows a trace of albumin. No casts were found.

January 23. No change in the condition of the liver. There is movable dulness in the right flank. In the posterior axilla liver dulness begins at the 8th rib.

January 28. The patient sweats much at night. The most tender point on the right side is in the lumbar region just at the extremity of the 12th rib. It is especially tender when pressure is made upwards. There is distinct sense of fulness and resistance between the 9th and 11th ribs on the right side laterally. On percussion a high tympanitic metallic note bounded below by the area of dulness which appears to be undoubtedly hepatic. The patient was punctured in the 10th interspace and there was found a small amount of pus. The abdomen is a little

full, everywhere soft. Towards the right flank the finger can be pressed well towards the renal region with perhaps a slight sensation of increased resistance. On bimanual palpation there is certainly great resistance below the costal margin and below the last rib, especially below the joint of the rib. Here there is also marked tenderness. From behind there is distinct fulness in the lower costal region on the right side and the intercostal spaces are not so well marked. The percussion resonance extends to the 10th dorsal vertebra on the right side. There is no distinct tenderness in the right lumbar spaces behind. Liver dulness in the mid-sternum is of three fingers' breadths in the nipple line from the lower margin of the 5th to the costal border in the mid-axillary line. Pulmonary note extends to the lower margin of the 6th rib. On percussion down in the nipple line liver dulness is uniform in the mid-axillary line. Liver dulness is distinct for a finger's breadth and there is the most metallic tympany extending from exactly the 8th rib in the mid-axilla to the upper margin of the 11th where it passes insensibly into the bowel tympany. There does appear, however, to be a slight dulness before the bowel tympany is reached. Anteriorly the metallic tympany extends to within 1½ inches of the nipple line. It extends to the posterior axillary line. When turned on the side the percussion at the posterior axillary line is distinctly flatter, although tympany extends.

February 2. The tympanitic note is even more intense than formerly. It is somewhat amphoric in character and extends to the right as far as the nipple line where it is distant from the nipple 10 cm. The area is triangular in shape, the apex being toward the sternum. The liver dulness extends far over into the left hypochondrium. Temperature ranges irregularly from 100° to 103°.

Transferred to the surgical side January 29, 1890.

Discharged April 12, 1890.

Surg. No. 304.

The patient was transferred to the surgical side for operation with a diagnosis of pyopneumothorax subphrenicus. The pain is in the right hepatic region and is localized. The physical signs of an area containing the pus cavity are present in the right axillary line about the level of the 9th to the 12th ribs. Temperature has varied between 100° and 103.4°. The urine shows a faint trace of albumin and a few casts.

Operation January 29. Under ether an incision was made in the axillary line downwards and inwards for four inches. About 2 cm. of the rib was excised. A large cavity was opened from which there spurted a thick mucus-like, somewhat sanguinous pus having the odor of H_2S. The cavity contained by estimation more than a pint of this. Except for that which spurted out at the time of incision there was a very small flow from the wound on account of the thick consistency of the pus. On palpation with the finger trabeculæ could be felt in all directions. Nothing of the intestine could be felt but above the wall of the cavity there is more consistence and what was thought to be the liver. The inner wall of the

cavity could not be reached with the finger. A portion of the contents were washed out with a weak solution of carbolic acid, but was later changed for plain warm water. The cavity was packed loosely with iodoform gauze and the wound dressed.

January 30. The temperature since operation has not been higher than 99.4.

January 31. On redressing some more of the discharge was removed and the cavity irrigated.

February 2. The wound was irrigated and redressed. There is still considerable discharge of the same character. An area of amphoric resonance is found just a little within the mammillary line and begins 10 cm. below the nipple. It is about 15 cm. in vertical diameter and 20 cm. in horizontal diameter. The opening seems to be just at the outer inferior angle. The liver is apparently pushed far over to the left side but hardly at all further down than normal.

February 9. The patient is doing well. The resonant area extends much lower down even to the crest of the ileum. The upper limit of resonance is now at the lower border of the 8th rib. The cavity has contracted considerably. The patient improved constantly and was discharged cured April 12. The temperature from the day of the operation was for the most of the time normal.

Treatment.—Alexander Monro [21 23] claims to have been the first to propose operation for an empyema of air in the pleura. Sprengel makes this statement. Bell says that Monro advised operation in his lectures. In his pamphlet,[23] in which he calls Hewson to account for borrowing this idea, he does not claim, so far as I could find, to have published this idea. It is said that Monro's operation was performed by Gooch [22] and Kellie.[30]

Hewson,[24] however, seems the first to have attracted notice with this opinion.

Benjamin Bell [34] is said to have advocated an aspiration syringe, and Bromfield gives an interesting item on the value of pulmonary gymnastics. John Bell [35] advised free incision to allow the lung to remain quiet and ease the other lung of any intrapleural pressure. It is seen that for the older surgeons traumatic pneumothorax at least had little fears.

Laennec, however, believed symptomatic treatment alone indicated an operation only when danger from suffocation is imminent, but Laennec was interested in that discouraging group of tuberculous cases which had hitherto, for the most part, escaped notice.

Until comparatively few years pneumothorax was considered a hope-

less condition, but during the past very few years nearly every article on this disease contains notes on its treatment.

Roser [195] advised the production of an open pneumothorax as preferable to an extensive, dangerous, plastic operation to obliterate the pleural cavity, and the production of pneumothorax for a therapeutic measure has been sporadically proposed and used in tuberculous cases, showing that the condition itself was little feared; and the method of Potain,[218] who introduced sterile air as an effusion was aspirated, thus avoiding the changes of pressure incident to the removal of a large amount of fluid, is worthy of consideration (Rodet and Nicolas,[230] Phalip,[322] Bruen [214]).

A group of cases the treatment of which has aroused great discussion are the suffocative or "valvular" ones. For some of these left alone would progress rapidly to death. v. Ziemssen [189] advised repeated capillary puncture to relieve the pressure, and Béclère's simple apparatus is worthy of consideration. In such cases the opinions have varied between repeated puncture, a permanent cannula or pleurotomy (Sunderland,[243] Crowdy,[303] Klinitz,[290] Bouveret [211]). Cannulas have been suggested by Ollebar.[178] Alexander,[312] Unterricht,[266 268] have especially advocated operation in such cases. The case of Galliard [343] is an illustration, and that of Gould.[183]

The consensus of opinion seems to favor disregard of the air and treatment of the effusion. In case the dyspnœa is extreme, Béclère's apparatus is recommended. Aspiration is usually contraindicated, since there is danger of opening a partially closed fistula, of rupturing a superficial cavity or especially of tearing the lung because of some adhesion which, as the lung is forcibly expanded, may tear the pleura. Hence the fluid may be allowed to escape as long as it will, but to subject it to a strong negative tension is a procedure of doubtful value. In this connection may be mentioned the danger of the production of pneumothorax in aspirating a simple pleurisy. There are two methods in use; one by aspiration, and the negative pressure which the ambitious clinical clerk will produce with a good pump is pretty formidable, and to exhaust all the fluid possible; the other is to slowly remove a small amount, about one liter; we have heard Naunyn especially emphasize this limit; and use the suction pump if necessary, but only cautiously. On the other hand, the value of extensive aspiration cannot be doubted and the air, if it does enter in a few cases, seems hardly to complicate the matter, or at least very little, and is of so

infrequent occurrence that it hardly should militate against the use of thorough aspiration in cases of simple pleurisy.

For the treatment of such cases, see Léger,[304] Lieutand,[303] du Bovis.[275]

If, however, pus is present the case should be treated as empyema, and pleurotomy at once performed. West[297] especially, and in the face of considerable opposition, taught this. In the case of an external pyopneumothorax the Birlan syphon drain is recommended by many (Aron[358]).

The gas seldom needs aspiration, yet Gould's[183] case is of interest. Also see Hopkins'.[309]

Antiseptic irrigations have been used, dilute salicylic acid (Senator), iodine solution (Potain), and Fütterer[264] recommended a procedure which others do not mention, of inserting a cannula at the top of the chest that air may escape, and then injecting a mixture of oil of cloves and water through another cannula until the chest is full.

The value of prophylactic measures is mentioned by Finley,[320] who attributed their observance to Thomson.

In these chapters we have endeavored to give a review of the literature on pneumothorax in such a manner that the reader may get an idea of the cases observed and the work done from nearly the words of the individual writers. Our cases of pneumothorax, several of them rare and many of them interesting, are reported somewhat at length in the hope that one studying critically may not fail because of our omissions to get the best possible view of the case.

In the chapter on the mechanics of pneumothorax we have tried to give a fair idea of the consensus of opinion on various much disputed and by no means settled topics. Should anyone desire to examine the experimental work which we have done in this field, we would call his attention to the paragraphs on Gas Analysis, the Distension of the Affected Side, and the Cohesion of the Pleuræ. To the work which was purely confirmatory of that of previous writers, there are mere references in the various chapters.

In conclusion, I wish to acknowledge my obligations to Dr. Osler, at whose suggestion and under whose inspiration this paper has been written, and the cases of whose clinic I am privileged to report; to Dr. Halsted, who placed the surgical records at my disposal; to Dr. Howell and to Dr. Mall, in whose laboratories I have worked and from whose suggestions I have profited.

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CLINICAL OBSERVATIONS ON BLOOD PRESSURE

CLINICAL OBSERVATIONS ON BLOOD PRESSURE.

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II. Method of Observation Followed.

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I. Introduction and Historical.

The physiology of the present day, so far as it concerns itself with the cardiac, vascular and nervous apparatus of the blood-circulation, may be said to be absolutely dependent on the kymograph as its instrument of precision. A Harvey could discover the broad fact of the circulation itself without any such mechanical aid. But the general outlines once drawn, the fundamental principles once recognized on the basis of the more obvious, almost anatomical phenomena of the heart and vessels, it was necessary for those who were to fill in the details of a great science to become more and more precise in their technique, and for different workers to adopt an accurately determinable mathematico-physical basis of comparison for their results. The determination of the hydraulic pressure of the moving blood within the vessels afforded a means for this comparison in all studies of the force of the blood-propelling mechanism. Stephen Hales in the middle of the eighteenth century was the first to accomplish this determination in experimental animals, his results being expressed in terms of the weight of the blood itself. Hales's methods were of the simplest imaginable, and it was not until Poiseuille (1) in 1828 introduced the mercury manometer as a measuring instrument, and devised a means of preventing the coagulation of the blood within the apparatus, that approximately continuous observations of blood pressure could be made, even in the laboratory. Ludwig (2) in 1847 made the manometer self-registering, an accomplishment which "has marked an epoch, not only in the study of the circulation, but in the whole science of physiology" (W. T. Porter). Since the introduction of Ludwig's kymograph, hundreds of experimenters have applied themselves to the problem of more accurately registering the state of the pressure within the arteries and veins, the capillaries, and the chambers of the heart itself. The number and endless variety of the instruments devised for these purposes may be seen from a glance at the earlier papers of Vaschide and Lahy (3).

The study of blood pressure in the physiological laboratory having proven vastly instructive as to the processes of the circulation, it was not long before attempts were made to accomplish similar studies in man, hampered as such endeavors were by the radical impossibility of measurement by the methods applicable in animals, which necessitate direct access to the blood within the vessels. As early as 1855

Vierordt (4) suggested the principle of the indirect estimation of the pressure of the blood in man by means of the counter-pressure necessary to cause disappearance of pulsation in a peripheral artery—a principle that recurs in most modern sphygmomanometers. Forster and Béhier in 1868 endeavored to apply the principle of Marey's sphygmograph to the measurement of the arterial tension at the bedside, and Landois and Philadelphien estimated this tension in terms of the actual weight necessary to produce collapse of the walls of a peripheral vessel. Unsatisfactory and faulty in theory as it appears to-day to attempt the determination of the tension within an elastic tube of varying length and calibre by means of local counter-pressure applied through a solid block, it remained the only method in use until v. Basch (5) in 1876 contrived to give in terms of millimeters of mercury the amount of pressure necessary to occlude the lumen of the radial artery. The new principles evolved by v. Basch were the compression of the artery by means of a bag, or pelotte, containing fluid, and the use of terms of measurement which could be compared more or less directly with the figures of the physiologist. For the first time it was now possible to obtain even approximately accurate or useful clinical information from clinical blood pressure measurements. Potain (6) in 1889 made use of air instead of a fluid medium to transmit pressure to the compressed artery, and measured the pressure by means of a manometer constructed on the aneroid principle, but graduated in terms of millimeters of mercury.

Though each of these instruments successively marked an advance in the perfecting of a means for estimating arterial tension in man, none overcame the mechanical difficulties involved in applying a measurable pressure to the peripheral arteries by a device sufficiently easy or accurate in every-day use to make them of extended practical value. Each attained a certain limited popularity for a time, especially on the Continent, and undoubtedly stimulated interest in this important field of research, besides giving many valuable indications of circulatory phenomena, but it was not until a new principle of artery compression was devised that the way was open to the general adoption of the sphygmomanometer, and the recognition of its utility in the clinical field (7). The method of artery compression from above downward involved all the mechanical errors that make finger palpation so unsatisfactory, rendering such factors as size of artery, depth below the surface, and the relation of surrounding tissues important sources of

inaccuracy. Tigerstedt (8) estimates the probable error in the use of the v. Basch instrument as 30 to 80 mm. Hg., while the principle of gaseous transmission of pressure as used by Potain must produce a still wider range of variation for the figures so obtained, from actual intravascular blood pressure. The newer principle eliminates these difficulties in great part, and according to Janeway (9), removes the "largest source of error in the clinical estimation of blood pressure." It is embodied in three separate forms of apparatus, all of which have already obtained rather wide clinical usage; the Hill-Barnard, the Gärtner, and the Riva-Rocci sphygmomanometers. Instead of from above downward, the pressure is applied to the artery equally in all directions, perpendicular to its wall at every point, through the medium of the surrounding tissues, by means of a hollow extensile rubber band which encircles the extremity of the patient. This band is connected by a continuous closed air system of rubber tubing with a bulb for increasing the pressure and with a manometer for registering it. By the law of gases equal pressure exists in every portion of such a system.

The application of this principle differs widely in each of the three instruments. In Gärtner's tonometer the pressure is applied to a digital artery and the maximum arterial pressure estimated by measuring the force just necessary to keep the artery occluded. The tip of the finger is blanched and then the blood is prevented from returning by pressure applied around the finger between the first and second joints. This pressure is gradually lowered until flushing under the nail indicates that the lumen of the artery is restored, and that the systolic blood pressure in that artery has just overcome the pressure exerted by the instrument and registered by the manometer. To confirm a first reading the finger must again be blanched, thus requiring practically a complete repetition of the entire process. Though this idea is extremely ingenious and the use of the apparatus in selected cases may give accurate results, its practical defects are many. In the first place the application is not always easy. With the co-operation of a suitable patient it is not usually difficult to obtain a satisfactory blanching of the finger tip, but with a toxic or anæsthetized patient we have found it extremely unhandy, so much so as to preclude its routine use in following consecutive pressures repeated at short intervals in these conditions. Furthermore, the small digital arteries are subject to great local vasomotor changes even in health, but

especially in disease, which render them unsuitable for estimations of this kind. In clinical conditions where the arterial tension departs most from the normal and where we most need accurate blood pressure observations the Gärtner tonometer is untrustworthy. Gärtner himself (10) attempts no estimation under 70 mm. and others have found the lower limit of accuracy much higher; while its upper limit of 260 mm. is far below that of many cases of hypertension. We have found it impossible of application in infants and unsatisfactory in the negro.

The Hill-Barnard instrument is generally supposed to measure the "mean" arterial blood pressure, and is much more complicated in principle than Gärtner's apparatus. The constricting hollow band is placed around the upper arm and is connected with a delicate metal tambour. As pressure is increased in the air system a needle attached to the tambour describes oscillations corresponding to each cardiac systole, and which reach a maximum when the external pressure is at a point corresponding to the "minimum" blood pressure. This principle, first proposed by Marey, and made use of in numerous other sphygmomanometers, such as the Mosso, Hill-Barnard, Erlanger, etc., is usually supposed to determine the mean arterial pressure, but, as pointed out by Howell and Brush (11), actually measures the minimum or diastolic pressure. This instrument requires practice and skill in handling and considerable experience in adjustment, so that it is unsuited to general clinical usage. Besides, it necessitates the use of a metal tambour, which is always subject to errors of adjustment. Janeway, and Howell and Brush, have examined carefully and critically into its practical value and unite in declaring it both unsatisfactory and inaccurate. Janeway states that, at the time of writing, his Hill-Barnard apparatus registered 26 mm. out of the way. The possibility of this error in adjustment makes observations on variations in blood pressure by such an instrument valueless, unless controlled carefully by some absolute standard. This standard is found in a mercury manometer, where the height of the column cannot vary with equal pressures except for the fraction of a millimeter due to thermic changes.

The third instrument, the Riva Rocci, like the Gärtner, is primarily intended to measure maximum pressures, but the pressure is applied to the brachial artery and disappearance of the pulse at the wrist is taken as the signal of its occlusion. This obviates the errors and difficulties of the Gärtner method.

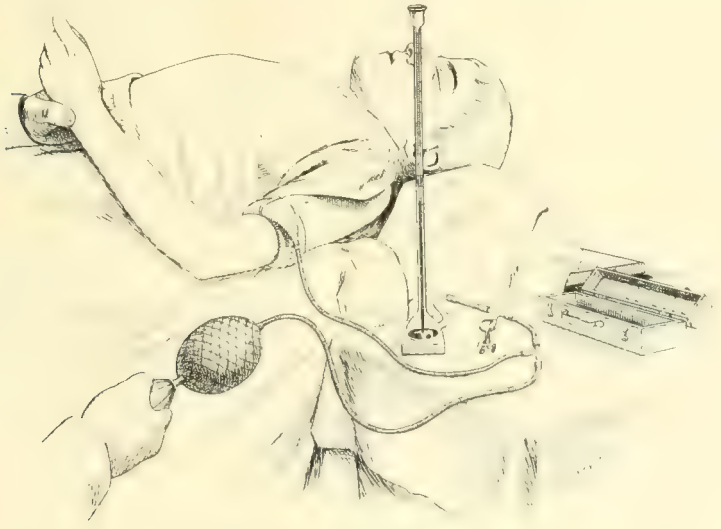
The ideal sphygmomanometer for use in man, if the physiological point of view alone is considered, is the instrument recently perfected by Erlanger (12), which combines the principles of the Riva-Rocci and the Hill-Barnard, and so gives both maximum and minimum and hence mean pressure measurements. But unfortunately the amount of apparatus necessary makes this instrument too bulky and complicated even for routine hospital use, and is of course out of the question for determinations in outside practice, hence its function in practical medicine will probably be that of an aid to the study of conditions rather than of cases. While in physiological research and occasionally clinically it is of interest to know the strength of the pulse at every point in its wave, such refinement of observation is confusing and unnecessary in general practical usage, as Howell and Brush have shown that even under extremes of cardiac or vascular changes the general trend of arterial tension is given by instruments measuring either systolic or diastolic pressures, so that practically an instrument simple enough in construction to be easily portable, which will measure with reliable accuracy the arterial pressure at any one phase of the pulse wave, will be the one whose clinical use will be the widest and whose indications will be the most generally consulted.

It is essential for the most satisfactory development of the blood pressure work that some standard should be generally recognized, and that the present confusing methods be obviated whereby observations are being reported indifferently in terms of mean, minimum and maximum pressure, and by various instruments, so that the results appear conflicting and unreliable. As the Riva-Rocci principle is the simplest and admits of the most practical and accurate application it seems justifiable to urge that maximum pressure as determined by it should be adopted as this standard. In its original form the Riva-Rocci sphygmomanometer was somewhat unhandy for clinical use and various modifications have suggested themselves in the endeavor to permit it a wider field of usefulness. Stanton (13) has recently described a form of apparatus which he has used in hospital work, but the complicated arm-piece, bulky foot pump and the form of the manometer make a handier instrument desirable.

Eimer and Amend are making a simpler modification after a model furnished by one of the authors, and this we have found compact, easily portable and accurate (14) throughout a year's use, in a great number of cases studied in the wards of the Thomas Wilson Sani-

tarium and the Johns Hopkins Hospital, showing every degree of arterial tension from 40 to 50 mm. Hg. in ill children, hæmorrhage, and profound traumatic and surgical shock, to 350 mm. Hg. and over in intracranial hæmorrhage and uræmia.

The accompanying cut illustrates the apparatus we have used. The rubber arm-piece is enclosed in canvas, with hook and eye attachments which allow of its being fitted to any arm from that of an infant to that of a large adult. The valve and reservoir bulb are similar to



Modified Riva-Rocci Sphygmomanometer.

those used in the Paquelin cautery. The mercury manometer is simplified and the upright tube is joined in the middle so as to fit into a small, easily portable case, making the instrument suitable for use in general practice. The arm-piece is placed near the middle of the upper arm by preference and adjusted to fit snugly. The pressure is then raised in the enclosed air system by means of the pressure bulbs until the pulse disappears at the wrist, this point is noted and the column of mercury allowed to fall until the pulse returns. The position of these points is then verified by alternately raising and lowering the pressure by squeezing the reservoir bulb, and the mean between them is taken as the maximum arterial blood pressure. A reading within 2 or 3 mm. is considered satisfactory. To make

and confirm an observation in this way requires less than a half minute. In cases where we have wished to follow the pressure at short intervals, for example during labor pains, we have been able to make them easily every half minute for an hour or more consecutively.

Though this instrument is intended primarily to give systolic pressures, the minimum pressure may be obtained by it with fair accuracy when a rigid system of tubing is used, by the method Gumprecht (15) describes, and which depends on the same principle as that of the Hill-Barnard instrument, i. e. observation of the point at which the mercury column makes its maximum oscillations after the tude leading to the reservoir bulb is shut off. Determination of diastolic pressures by this method, however, is not capable of as high a degree of precision as by the use of Erlanger's more elaborate instrument, and is only possible at all in a minority of cases.

II. Methods of Observation Employed.

We have invariably used the upper arm as the place of choice for applying pressure, and the radial artery for palpation. The band is placed preferably midway between the elbow and shoulder, as here we avoid the condyles of the humerus and in muscular men the sharp slant below the bulging of the biceps. However, any place above the condyles is suitable. At the beginning of our observations we supposed that the position of the band and the thickness of the arm would, as seems probable theoretically, affect the readings, but we have come to regard these factors as of little importance. We have so repeatedly found stout men otherwise apparently normal with a measured blood pressure of 120 to 130 mm. Hg., that we look upon the size of the subject's arm as a very small factor, except in extreme grades of emaciation, where the absence of the cushion of tissue makes equal compression difficult and tends to give a higher reading.

The conclusions here given are correlated from observations taken in the operating rooms and wards of the Johns Hopkins Hospital and the wards of the Thomas Wilson Sanitarium. No single series of observations on one case has been regarded as justifying a conclusion, but each was controlled by further observation and repeated experiment. Thus any individual chart here inserted, though an exact copy as taken at the bedside, is merely given as typical of many others and should not be regarded as an unconfirmed record. Many highly suggestive observations we have excluded entirely, because we have not

had the opportunity of verifying and controlling them by further study of similar cases. In watching the effect of any single treatment, either drug or mechanical, we have taken repeated observations at short intervals of 2 to 5 minutes, for some time before the treatment was begun to obtain a comparative base line, during the treatment, and afterwards until its effect ceased.

All the observations were taken by one or other of the authors, and when we have followed a case consecutively for long periods, such as 24 to 48 hours or more in succession, we have relieved each other alternately, one remaining constantly at the bedside.

We have always attempted to avoid possible extraneous sources of error such as excitement or muscular exertion on the patient's part. We have found, however, that while these factors are important physiological sources of variation in blood pressure in normal individuals, in patients surrounded by the ordinary precautions of the sick room they but seldom interfere. We are coming more and more to believe that blood pressure is a more constant quantity in health and a more absolute indication in disease than is generally supposed. We have attempted elsewhere to point out the value which correct estimates of arterial tension must be to the clinician to supplement a knowledge of pulse rate, temperature, general condition and the indefinite palpation estimate of pulse force. Already throughout the medical world the rapidly increasing interest in blood pressure work sufficiently evinces the general understanding of its practical value, and we feel convinced that the time is not distant when the careful synopsis of every case will contain precise determinations of arterial tension, and that both the diagnosis and treatment of many conditions will be more intelligent through accurate knowledge of this important factor in the circulating mechanism.

Up to the past few years, and before the recent improvements in the mechanical determination of arterial tension had been effected, all conclusions as to the actual effects of treatment upon this important symptom in disease had to be deduced either from physiological experiment or from palpation judgments of variation in tension. The manifold errors inherent in such indefinite methods are too evident to need emphasizing. When a mathematically accurate means of determining arterial tension at the bedside is possible, judgments based on such faulty data as were previously alone obtainable can be accepted only where they agree with the results of more precise methods. For this

reason we have considered our results valuable, even though in part they contradict prevailing opinion. Besides confirming or correcting the broad generalizations governing the classification of drugs as stimulants, depressors, etc., accurate measurements of tension permit a further refinement and individualization in treatment than is possible by cruder methods. Thus, although palpation of the pulse and animal experimentation may give proper recognition to strychnia as a stimulant, they cannot accurately gauge the period or extent of its stimulant action in single cases of different nature.

In recording the observations on which this report is based we have invariably used the graphic chart method after Dr. Cushing's suggestion, with each blood pressure reading noting the pulse rate and where important the rate of respiration. We have our charts so scaled that a pulse rate of 80 and blood pressure of 130 mm. Hg. will fall on the same line, using this as a base line of approximately normal readings. By blood pressure determinations on a large number of practically normal individuals we have found that we may expect the following pressures:

For children, 75 to 90 mm. Hg. during the first 2 years and 90 to 110 mm. Hg. during early childhood, the determinations here being more absolute than in the adult, where organic cardio-vascular changes constitute a more frequent and important source of variation from the average norm.

For young adult males, 130 mm. Hg. in the dorsal decubitus. In general there is a slight gradual rise in blood pressure with increasing age, pari passu with, if not dependent upon, arterio-sclerotic changes.

Women usually have a blood pressure of 10 to 15 mm. Hg. lower than men, age and general condition being the same.

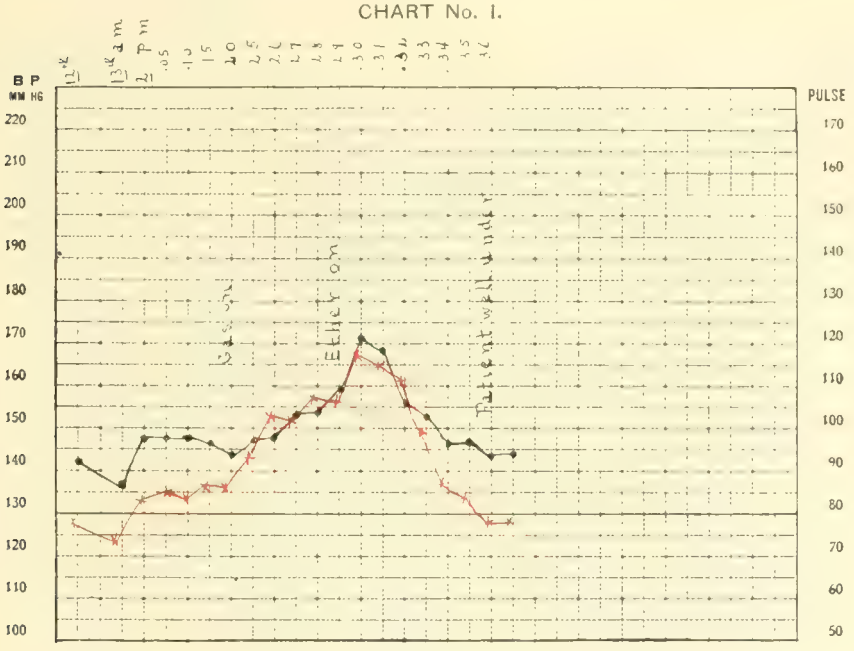
A sitting or standing position gives a higher reading by 5 to 10 mm. Hg., excluding the immediate effects of exertion.

We wish first to review some of the conditions in which we have found blood pressure observations of especial value or interest. Our studies have been particularly directed towards determining the actual measurable effects of the therapeutic agents in general use more or less directly intended to produce changes in arterial tension, and we present these results with our conclusions as to their respective value in more detail.

III. Blood Pressure Observations in Surgical Cases.

The most striking alterations in arterial tension which come under observation are perhaps met with on the operating table and in connection with the after-treatment of surgical cases.

CHART No. I.



Anæsthesia with Nitrous Oxide and Ether.

1. The Effects of Anæsthesia.

(1). *Ether.*—Ether causes a rise in blood pressure during the first stage of anæsthesia probably in response to the irritating effects of the vapor on the respiratory mucous membranes; and also during the second stage, this rise being apparently in great part due to muscular exertion and the accompanying general excitement. (See Charts I-IV.) When the factor of excitement is eliminated as we were able to eliminate it in experiments on ourselves, this rise is slight or absent. In the deeper stages of anæsthesia the pressure falls to a level slightly above the normal, or occasionally slightly below, especially when there is sweating, but never very low if uncomplicated by the later effects of

severe nervous insult, persistent asphyxia, or cardiac collapse. As the patient begins to come out from deep etherization the pressure generally rises. On the whole, ether seems a very safe general anæsthetic as judged by its effects on arterial tension, causing a transitory initial rise, a slight later fall, probably associated with perfect dilatation; but with no real depressant action on the circulatory mechanism, acting rather as a stimulant in the normal individual.

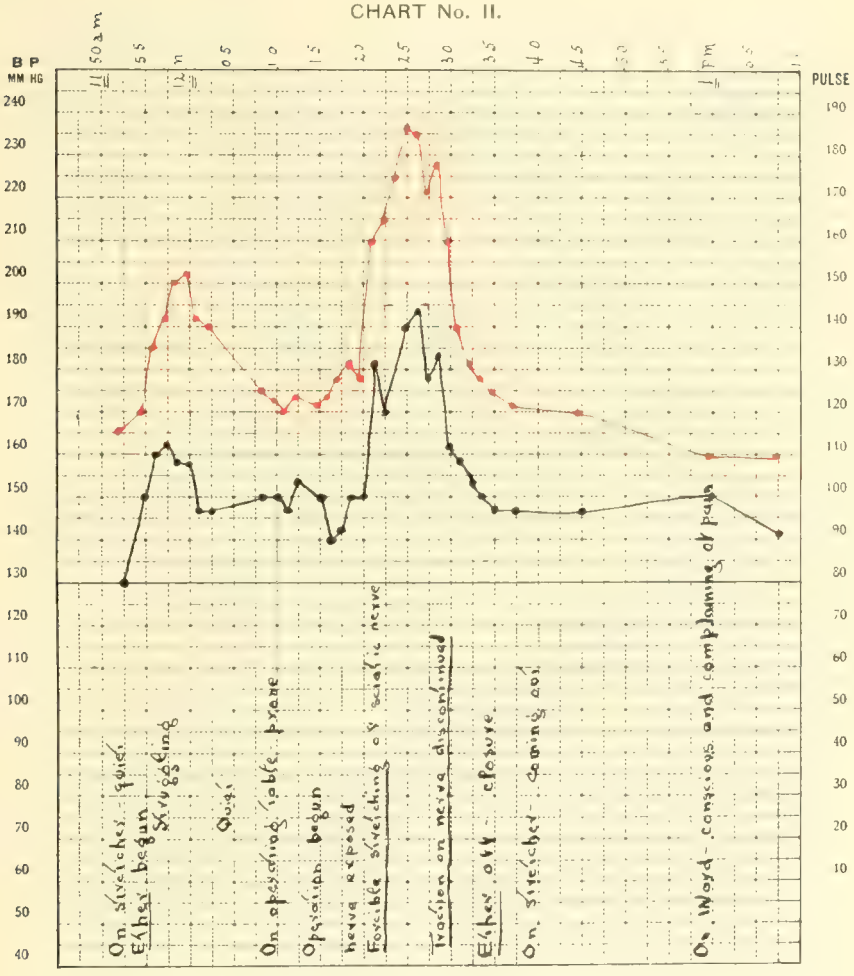
(2). *Nitrous Oxide.*—Anæsthesia with nitrous oxide is accompanied by a rise in blood pressure apparently not due to any true stimulant action but rather to the associated partial asphyxia. (Chart I.) Asphyxia is always associated with hypertension until the final stage of syncope and is, of course, always an element in nitrous oxide anæsthesia except the most temporary. When used as preliminary to ether anæsthesia there is a similar initial rise in tension to that seen when ether alone is used, but the later secondary stage of excitement with great rises in tension accompanying muscular exertion is almost completely eliminated, and the patient passes from primary to deep anæsthesia very rapidly, saving 5 to 10 minutes of exhaustive struggling with its attendant hypertension.

(3). *Chloroform.*—Chloroform, on the other hand, appears dangerous and uncertain when given on a mask. Its action is distinctly depressive from the start, the pressure failing definitely almost from the first whiff, and remaining uncomfortably low throughout. (Chart XIV.) Respiratory failure is not infrequent at any stage, apparently due either to a direct inhibitory action on the center, or to anæmization of the center secondary to vascular depression. The single exception to this seems to be in the case of pregnant women during labor, who take chloroform uniformly without bad effect. We have observed that a constant rise in blood pressure occurs during labor and this may account for the relative harmlessness of chloroform at this time, by counterbalancing in a measure its depressive effect.

2. The Effect of Operative Interference on Blood Pressure.

The effects of the operation itself are primarily pressor, that is, each successive step in cutting or manipulation produces a transitory rise in blood pressure analogous to the physiological response to pain stimuli. (Charts II-IV.) The patient, though anæsthetized, is receiving peripheral afferent impulses in the nervous centers, and may thus be said to be suffering subconscious pain. The ultimate

CHART No. II.



Forcible Stretching of Left Sciatic Nerve for Sciatica.

(1) Primary "ether rise" in blood pressure, with slight acceleration of the pulse rate.

(2) Marked rise in blood pressure, with greater acceleration of the pulse rate, during the manipulation of the nerve (transient pressor effect of peripheral nerve stimulation).

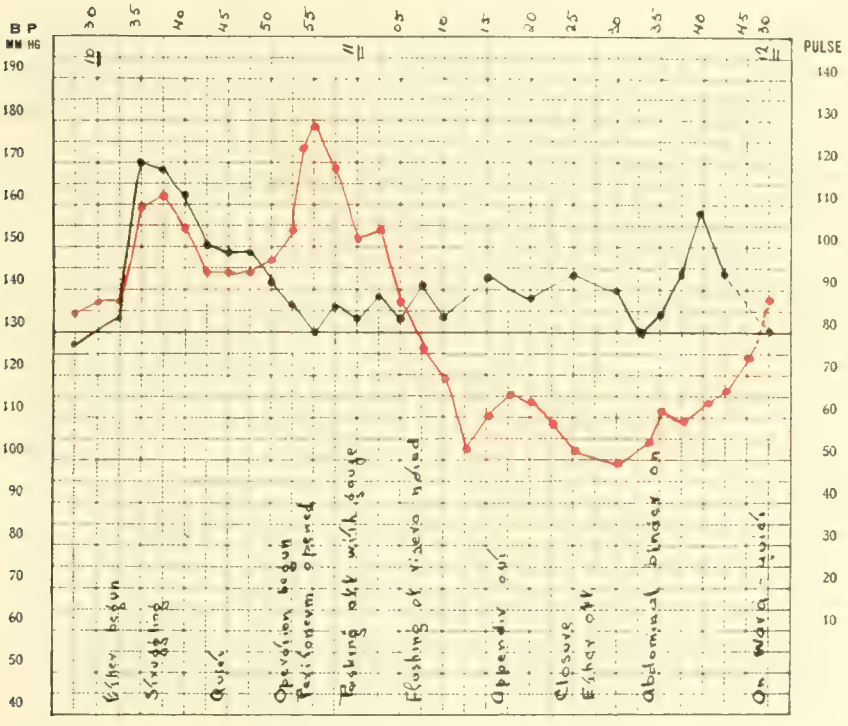
effect of such impulses if repeated beyond a certain limit seems to be the same whether the patient is able to feel them or not. Beyond this point the vasomotor system is unable to respond and further nervous insult produces a lowering of blood pressure. This drop in blood pressure may be either transitory or permanent. Many cases during some extensive operation involving great nervous irritation, especially in abdominal and brain surgery and the major amputations, show a marked fall in blood pressure (Charts III, V, VIII, XIX), which may only remain at a dangerous level for a few minutes, and then if the main operation is concluded, will gradually rise during closure to a safe level without special treatment. On the other hand, this initial fall in blood pressure may be the beginning of a vasomotor depression which under continued manipulation goes on to profound shock.

Crile's dictum, based upon physiological experiments, that a low blood pressure is the one essential feature in the production of the symptoms of shock, is abundantly substantiated by our observations, which permit us to go a step further in stating that it is not only the essential feature of shock, but that a fall in blood pressure is its primary manifestation and affords a surer and earlier warning of its imminence than any other symptom or sign. We believe that the only safe way of recognizing the early onset of serious depression during operation is to follow the blood pressure with some instrument that can be depended on for accurate readings. (Chart XI.) Pulse rate does not afford a true guide to vascular strength, for although a rapid pulse is usual in shock, the fact that it may be slow is well known. Bloodgood (16), in referring to a case of profound shock with a pulse rate of 80, says it is a "good illustration of a fact which we frequently observe, no marked acceleration of pulse rate in extreme conditions of shock, especially if there has not been great loss of blood." Conversely, great variations in pulse rate may occur under ether anæsthetization or in connection with great nervous excitement quite unassociated with variations in arterial tension.

With an accurate means at hand of recognizing the successive stages of operative vascular depression, we would advise a definite procedure of blood pressure control, varying, of course, with the individual requirements of each case, and with reference to the graphic blood pressure curve as the best basis for judgment. The approximate normal for each patient should be determined by one or more observations

before beginning the anæsthetic and if possible by others on the day previous to operation. After a marked fall below an approximate normal the anæsthetizer should feel warned of danger and keep closer

CHART No. III.



Laparotomy (Appendectomy).

(1) Initial etherization rise in blood pressure.

(2) Primary rise in blood pressure from peritoneal irritation.

(3) Secondary fall in blood pressure from continued peritoneal irritation coincident with and due to splanchnic engorgement, and without change in pulse rate. This curve is typical of all laparotomies, and is the analogue of that produced by intestinal perforation and peritonitis, only with a relative shortening of the pressor phase.

watch of the pulse and general condition and rectify any possible fault in the administration of the anæsthetic that might be responsible. If the pressure rises promptly spontaneously or with

stimulant, as it not infrequently does, he may feel justified in proceeding, though still on guard. If, however, the blood pressure continues to fall or remains without rise at a dangerous level, he should initiate active steps. The operator should be advised that the patient is not doing well and by a glance at the chart may find reason to expedite his manipulations according to the degree and rapidity of the depression. It is probably best in cases where respiratory distress is not a striking feature to continue the anæsthetic if ether is being used, as the small amount required to continue the narcosis is not as injurious as the consequences of coming out during the operation with the possible necessity of repeated anæsthetization. Crile has shown that a constant degree of narcosis has less tendency to produce shock than allowing the patient to come nearly out and then renewing the deep anæsthesia.

If the ether is withdrawn a moderate dose of morphia is advisable to control the restlessness which is so frequently distressing as consciousness returns, and which may aggravate any existent depression.

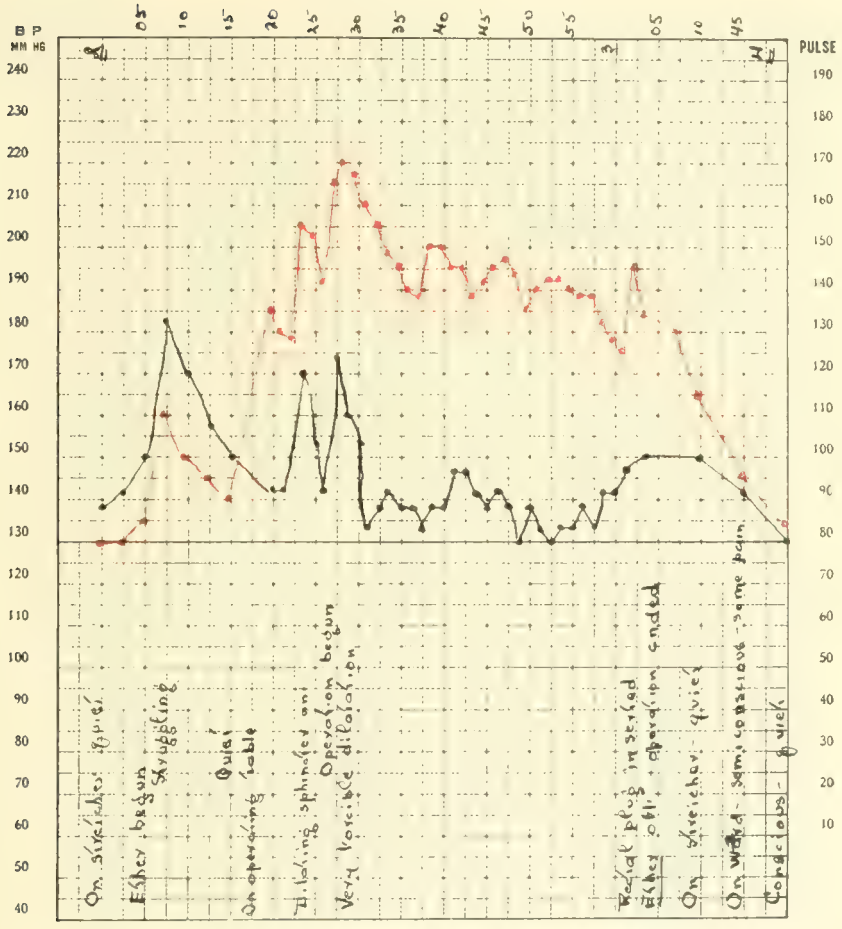
Chloroform should be withdrawn immediately at the onset of dangerous depression and if a further anæsthetic is required ether substituted.

The return of the arterial tension toward normal should be assisted by both mechanical and medicinal means. Inclining the patient with head down and feet up tends to raise the pressure by emptying the flooded splanchnic area into the general circulation. Abdominal compression as advised by Crile and Cushing acts in the same manner, and compression of the extremities as by strapping or using Crile's rubber breeches increases peripheral resistance. The respective value of the so-called stimulants will be detailed further on, but as there has been much recent discussion as to whether drugs which undoubtedly raise the blood pressure in other depressed conditions are also indicated in the rather unique shock complex, we give here the results of our observations in this condition, although they as yet number but ten cases studied in full.

3. Surgical and Traumatic Shock.

Of all well characterized and frequently observed clinical pictures, that described by the term "shock" has perhaps been the subject of the most varied and widely differing interpretations. No condition affords a greater mystery in the explanation of its more intimate

CHART No. IV.



Whitehead Operation for Hæmorrhoids.

(1) Primary "ether rise" in blood pressure.

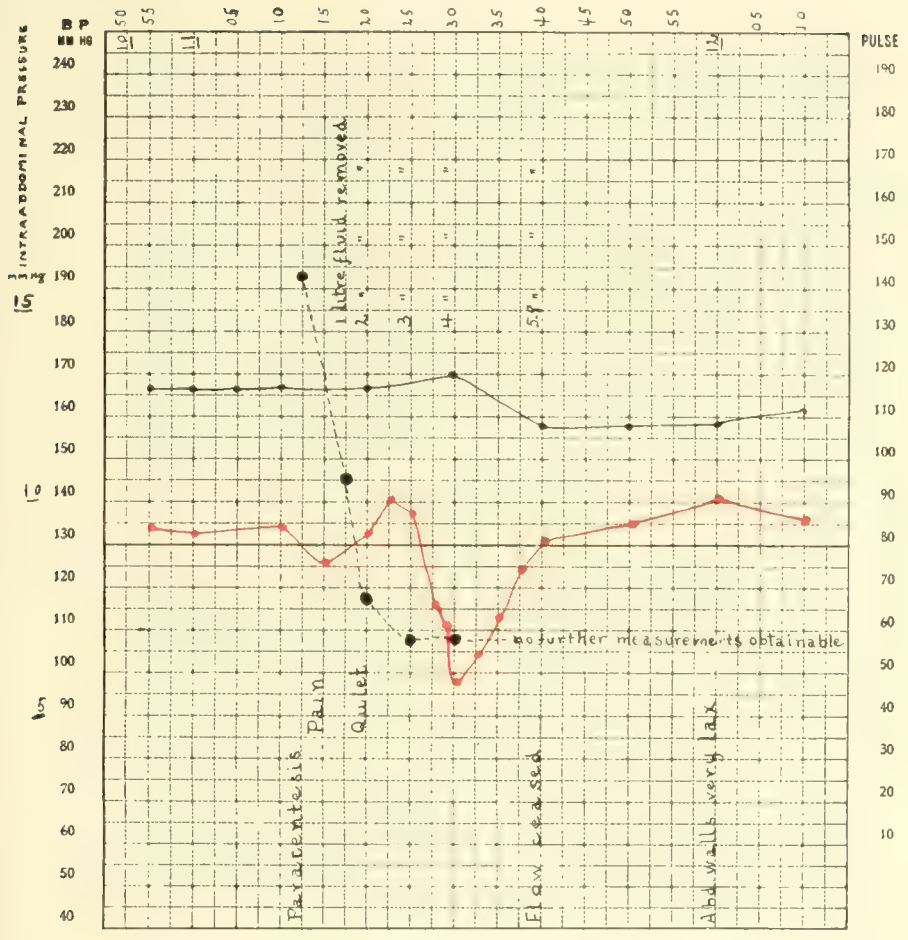
(2) Postural rise in blood pressure—abdominal compression by extreme flexion of the thighs in the position.

(3) Marked rise in blood pressure with forcible dilatation of the sphincter—peripheral reflex active throughout the operation.

causation, and though to-day this problem seems nearer to its final solution, still opinions are held by the most experienced observers, modifying theory and governing practice, that are as widely sundered as the poles. Just as no two theories of the origin of the symptom complex of shock are in agreement, so the principles of treatment in general repute, whether based on pure empiricism or derived in large part from the results of physiological experiment, differ as widely as is permitted by the limits of our pharmacopeia and the list of mechanical measures of therapy at our command. The tangle of contradictions afforded by the literature of shock is most discouraging to the reading student of the condition and calculated to produce in his mind the most disheartening uncertainty when confronted by the necessity of handling actual cases. It has seemed best to us, therefore, in attacking in a humble way one of the many sides of this apparently intricate problem, to discard as far as was permissible the uncertain and contradictory elements of mere opinion, however authoritative, and to set out from whatever of firm foothold could be found in demonstrated fact.

What is known with any degree of certainty about shock is this. The factors efficient in its production act by way of the peripheral afferent nerves on the central nervous axis. The resultant underlying condition, whatever it may be, which is responsible for the clinical or experimental phenomena of shock, affects chiefly the medullary vasomotor center, the periphery contributing only secondarily to the elaboration of the picture. As a consequence of this altered condition of the center, there is produced an abnormally low blood pressure within the whole arterial system, and to this marked hypotension are due all the features presented by the patient in shock. Further than this we cannot proceed safely without reverting to theoretical assumptions, whose value will be lessened in any case by the possibility of making other and contradictory assumptions of equal plausibility. There is no ambiguity possible in regard to two of the principles just cited, that shock is produced by the action of afferent impulses on the vasomotor center, and that the result of this action is a profound and dangerous depression of the general arterial blood pressure. It is in relation to the intermediate clause that doubt is still possible. The whole problem of the etiology and therapy of shock can be said to center around the question as to what the exact condition of the vasomotor center may be when peripheral stimuli have

CHART No. V.



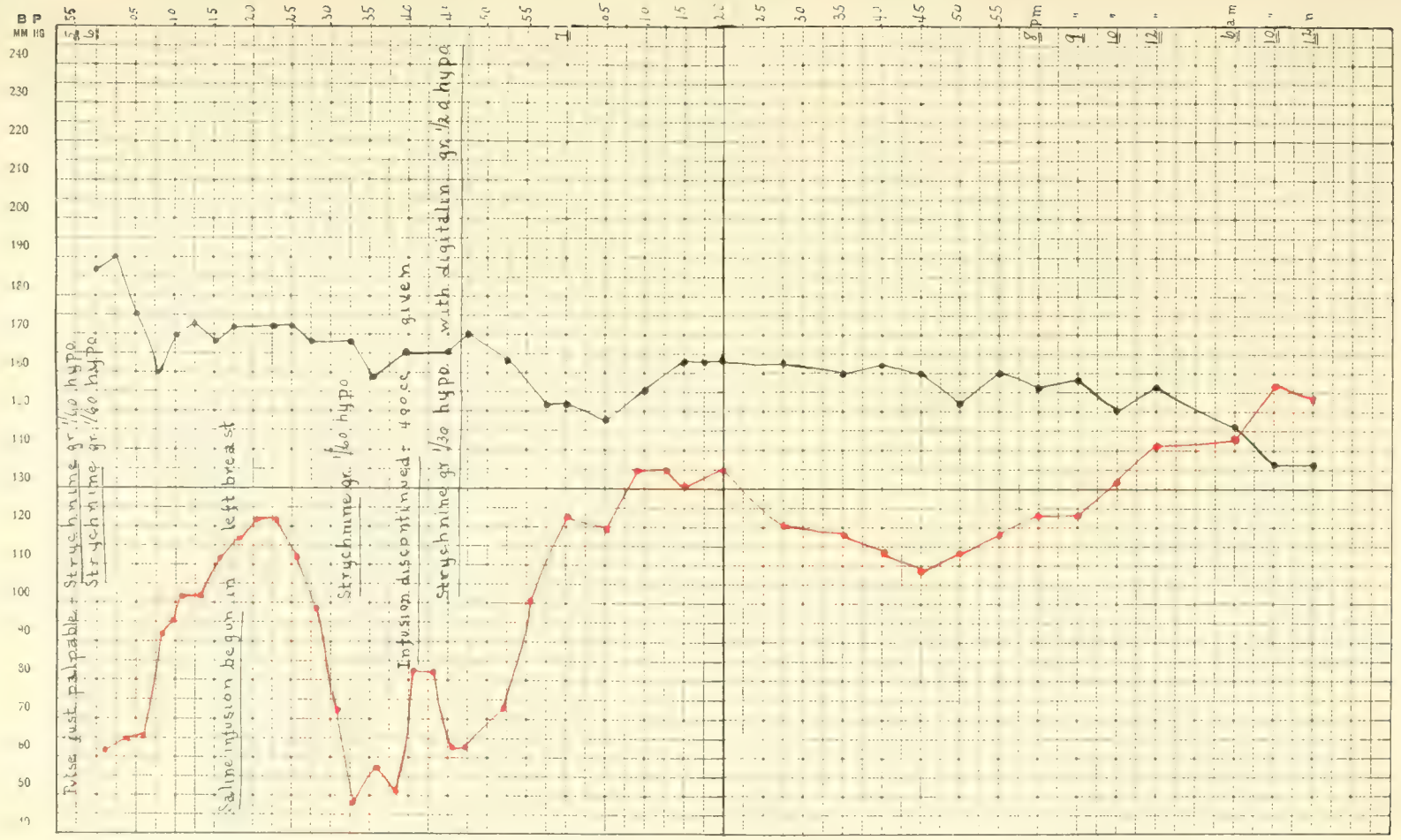
Abdominal Paracentesis.

The relief of intra-abdominal tension causes vaso-dilatation, and a fall in the systemic blood pressure, through enlargement of the capacity of the splanchnic blood reservoir. The fall in blood pressure is not exactly coincident with the fall in intra-peritoneal pressure—is not due to a gradual relaxation of vessels pari passu with the diminution of pressure upon them, but to a more sudden and general dilatation of reflex central origin. The vasomotor center being normally active, proper vascular tone is regained as soon as that center is stimulated to activity by the insufficiency of the circulatory pressure—and the normal blood pressure is restored by means of (splanchnic) compensatory vaso-constriction. This restoration of normal blood pressure is not due to abdominal muscular contraction and passive splanchnic compression, being complete long before the abdominal walls have regained their tone.

resulted in the characteristic lowering of the pulse tension. If that condition be one of exhaustion, if the center has been fatigued and the materials necessary for the continuance of its display of energy have been used up, by the mere excess of the stimuli received from without, then the treatment of shock by internal stimulant medication is equivalent to "flogging the tired horse to death"; and it is certain in every case of true shock not only to be without favorable result, but to deepen the already existent depression and to hasten and assure the fatal issue. If, on the other hand, the vasomotor center be not exhausted by excess of peripheral stimuli, if its activity has been but suspended or diminished as a result more of the character and mode of introduction of these stimuli, than of their mere number or degree, then no such necessary and absolute prohibition can be pronounced against the use of stimulant drugs and measures designed to affect the medulla in cases of profound or moderate shock. The point of theory at issue is as to whether the activity of the vasomotor center in shock is completely exhausted, i. e., whether the material necessary for its katabolic display of energy has been entirely used up, or whether that activity has been merely inhibited, and the material basis of power rendered unavailable, rather than totally consumed. The question in practice translates itself into the choice of the area for stimulation, excitant measures being properly applied only to the vasomotor periphery if there be actually central exhaustion, the center and the periphery together demanding stimulation if the central loss of control be not an expression of physiological exhaustion. In a word, are we to administer strychnine and digitalin, or should we withhold them in treating a case of shock? That the condition demands stimulation in some form for its certain relief, that the first duty of the attendant on a case of shock is to raise as quickly as possible the dangerously low blood pressure and to maintain it at a level compatible with life, is beyond dispute. In shock, as in no other morbid state, hypotension is not merely a symptom of value in diagnosis and prognosis, its degree is not solely a measure of the state of the circulation alone, but it is in itself the chief source of danger to life, and in too many cases the actual immediate cause of death. The lasting relief of hypotension by therapeutic measures means the radical cure of shock.

Stimulation simultaneously of both the vasomotor center and the peripheral vascular system by means of such drugs as strychnine, is in

CHART No. VI.


BP
MM HG
PULSE
Rise just palpable + Strychnine gr. 1/60 hypo
Strychnine gr. 1/60 hypo.
Saline infusion begun in left breast
Strychnine gr. 1/60 hypo
Infusion discontinued + 400 cc. given.
Strychnine gr 1/30 hypo with digitalin gr. 1/2 d hypo

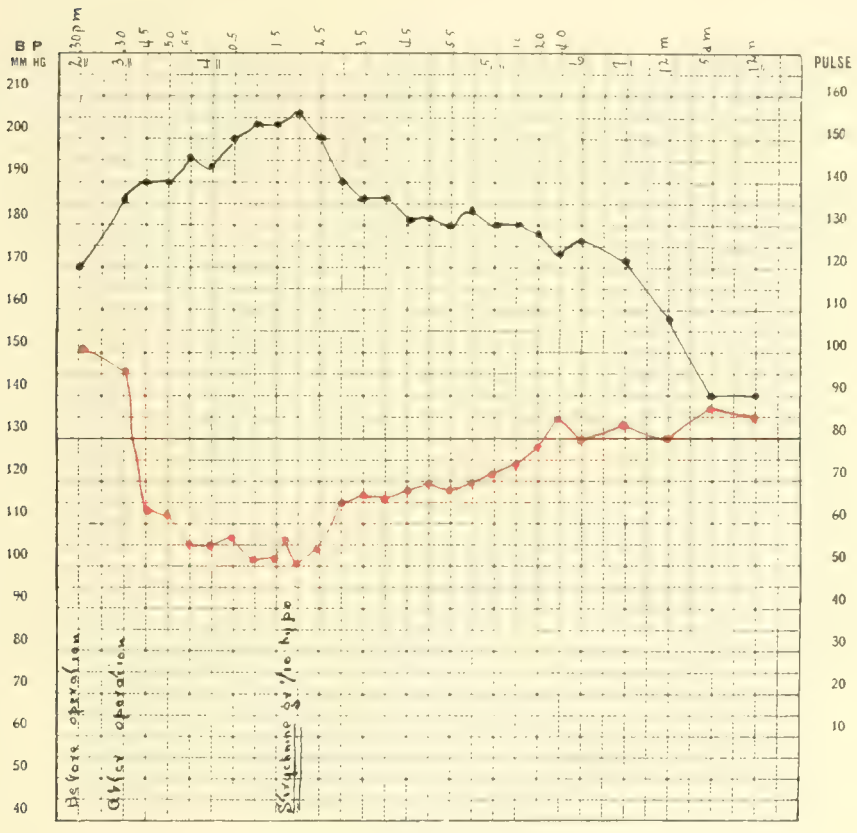
Traumatic Shock, without Hemorrhage. (1) Very low blood pressures marking the depth of the shock, which was otherwise clinically typical and marked. (2) Temporary rises in blood pressure after each of two smaller doses of strychnine. (3) Marked depression of blood pressure, with change for the worse in every symptom, during the continuance of an infusion of normal salt solution in the breast (considerable local tension). (4) After discontinuing the infusion, steady, progressive and permanent rise in blood pressure to the normal level, with complete recovery.

general the most rapid and most easily controllable means of raising the arterial blood pressure when it is below normal. As in the use of such measures their action on the nervous centers is the most important, we may for the moment leave out of account their effect on the periphery, and speak of strychnine, digitalin, and cocaine as "central vasomotor stimulants." If these drugs raised the arterial blood pressure solely by virtue of a direct action on the vessel walls and their terminal nerve filaments, there could be no doubt of the propriety of their use in shock. As their administration makes an additional call for the output of more energy by the vasomotor center, however, it is probable that if shock be merely the expression of exhaustion of that center, their total effect in shock would be to deepen the exhaustion and to hasten the final breakdown. It has been our intention, without regard to any theoretical views, to contribute something to the determination of this question clinically, and to discover the actual effect on patients in shock, of central vasomotor stimulation, thus defined.

In the present state of our knowledge we must describe shock as a state of depression of the function of the vasomotor center produced by peripheral nerve excitation unusual in kind or degree. Any more precise definition is not possible, and yet under it we must either broaden the application of the term "shock" beyond its usual acceptation, or else exclude arbitrarily from the category a condition that appears to correspond with all the items of the description used. There is a well-recognized group of cases characterized by extremely low arterial blood pressure, the result of trauma—differing only from shock, as generally understood, in the relative transiency of the vasomotor depression, and perhaps in the greater frequency with which they are associated with temporary loss of consciousness. Such a state of vasomotor loss of tone may be not infrequently seen following some injury or operative manipulation, when the afferent impulses excited have been considered to be of minor degree. The frequency of its production will be surprising to any one making a number of consecutive blood pressure observations on patients undergoing surgical operations. Once the blood pressure in these cases has fallen to a low level, they present a picture in every way identical with that of classic "shock" of corresponding degree, the only difference being in their subsequent course, which is usually characterized by a prompt return of blood pressure to the normal, with relief of all symptoms.

This return may be hastened in every case by the use of central vasomotor stimulants. In certain instances, however, this return does not occur—with continuance of the depressing peripheral irritation the

CHART No. VII.



Post-operative Depression.

Combined shock and hæmorrhage in twin labor, after prolonged forcible attempts at instrumental delivery.

Strychnine in large amount producing immediate and permanent rise in blood pressure.

blood pressure remains low or continues to fall—and the patient passes into a condition that is diagnosticated "shock" by the most critical. Every degree and duration of hypotension may be

produced thus by traumatic influences, without the occurrence of large hæmorrhage or cardiac syncope, and without any determinable change in the character of the trauma. Crile (17) here makes a distinction, reserving the term "shock" for the more enduring cases of low blood pressure due to trauma, and considering the more transient states under the term "collapse." We must recognize that vasomotor depression of reflex peripheral origin may be either very transient or very prolonged, but in the two cases the differences, etiological and symptomatic, are those of degree and not of kind. We believe that in all these instances we are dealing with the same morbid entity, and that the cases of "collapse" are merely examples of incipient or abortive shock, with early removal of the exciting cause before the most profound alterations have been produced in the medullary center. The mode of onset and the course of these transient states of vascular depression strongly suggests that some other cause than fatigue or exhaustion, in the physiological sense, is responsible for their phenomena, and hence for those of undisputed shock. We do not wish, however, to draw any argument from these cases of early shock (or collapse), but merely to recognize them, and to emphasize the fact that the observations here considered have only to do with traumatic low blood pressure conditions of long duration. Any case studied that might plausibly be interpreted as one of "collapse" has been excluded from the list, and we will report only on the results of central stimulation in instances where the hypotension was marked and lasting, and all the other clinical features of "shock" were pronounced.

We undertook the study of the blood pressure in shock at Dr. Cushing's suggestion, and during the past year we have taken every opportunity offered to obtain continuous pulse records in this class of cases. At first our observations were purely passive, and we were content with obtaining measurements of the pulse tension as we found it, simply following the results of the routine therapy which had been prescribed by those in charge of the cases. In some instances stimulant drugs were employed, and the results of these first casual observations, together with suggestions derived from our study of stimulants in other conditions, led us to believe that this class of drugs was often markedly beneficial in many instances of true shock. We were thus led in one or two cases to suggest the use of central stimulants in doses larger than the ordinary, and were encouraged in our tentative

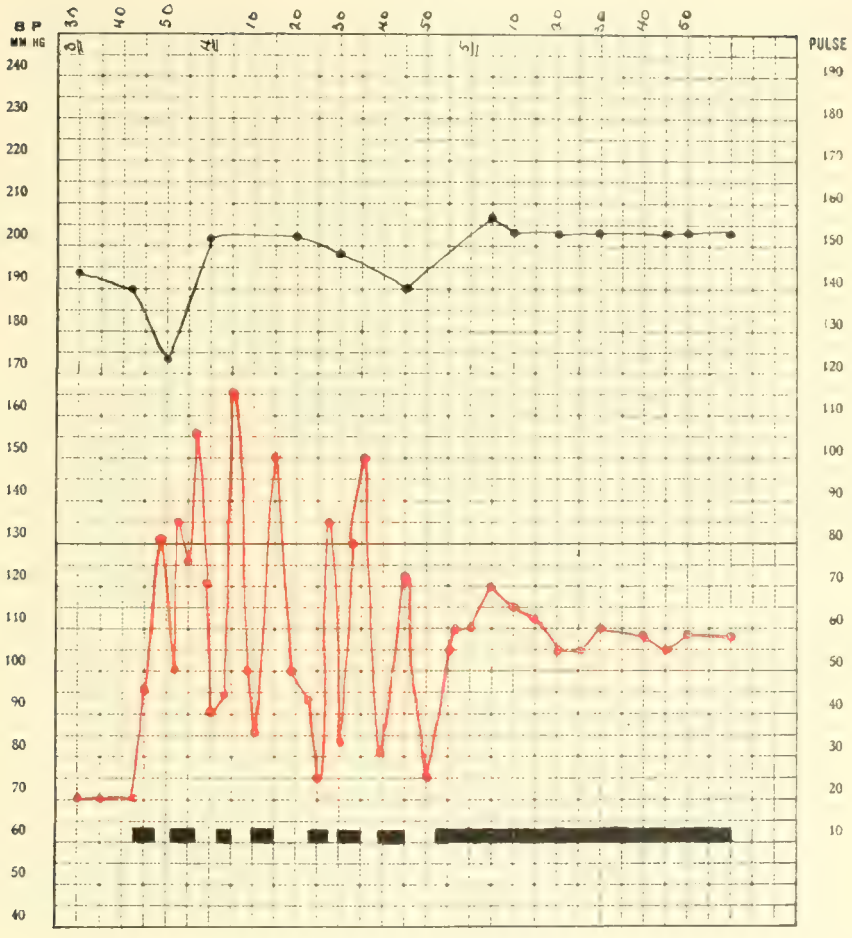
Fatal Gunshot Wound of Brain.—Elevation of Wagner bone flap and evacuation of sub dural blood clot. (1) Fall in blood pressure with relief of intracranial tension, from the previous compression level. (2) Low blood pressure when shock becomes manifest. (3) Slight and transient rise in blood pressure produced by strychnine and digitalin. (4) Adrenalin chloride injected intravenously producing great and immediate rise in blood pressure, with consequent hemorrhage from the wound, which proved the immediate cause of death. (Continuance of adrenalin transfusion indicated by continuity of black lines above.)

belief by the results obtained. Since that time, largely through the courtesy of Dr. Cushing and other members of the visiting and resident staff of the Johns Hopkins Hospital, we have been able to obtain continuous records of a series of ten cases of traumatic and postoperative shock in which treatment was guided largely by the course of the measured blood pressure, and drug stimulation was practiced under careful sphygmomanometric control. We have included in this series only perfect cases, that is, those only in which we procured complete blood pressure records without intermission from a period soon after or during the onset of shock, up to the time of death or eventual recovery. In every case the typical picture of shock was recognizable even without recourse to blood pressure measurements. In four cases only was there any great loss of blood, and in all of these the trauma and not the hæmorrhage was evidently the chief element in the production of the low blood pressure.

In the ten cases of shock in our series the drugs were used which we have uniformly found to raise the systolic blood pressure in all other conditions of hypotension. In all, strychnine was given, in seven cases digitalin was used also, and in one case (with positive effect) cocaine was employed. All the drugs were administered hypodermically. We have considered as a positive reaction under this treatment, a rise in blood pressure commensurate in degree and duration with that usually seen after similar doses in the hypotension of typhoid toxæmia. Such a reaction is held to indicate definite improvement in the patient's condition, the result of central stimulation (i. e., stimulation of the vasomotor periphery and center together), when it is not followed by a fall in blood pressure below its previous unstimulated level, unless that fall is again and finally corrected by still further stimulation (Crile, op. cit., p. 247). The smallest doses given were, for strychnine gr. 1-60, and for digitalin gr. 1-40; the largest, gr. 1-10 for each. In most of the cases the unit dose was a combination of 1-20 grain each of digitalin and strychnine, repeated as demanded by the course of the blood pressure.

To summarize our experience briefly: Of the ten cases, eight showed under central stimulation by means of strychnine, digitalin, and cocaine, a positive improvement as defined above. Of the eight cases improved, in five (or 50 per cent of the whole number stimulated) the improvement could be described as permanent, and the blood pressure returned more nearly to the normal with each succes-

CHART No. IX.



Profound Traumatic Shock.

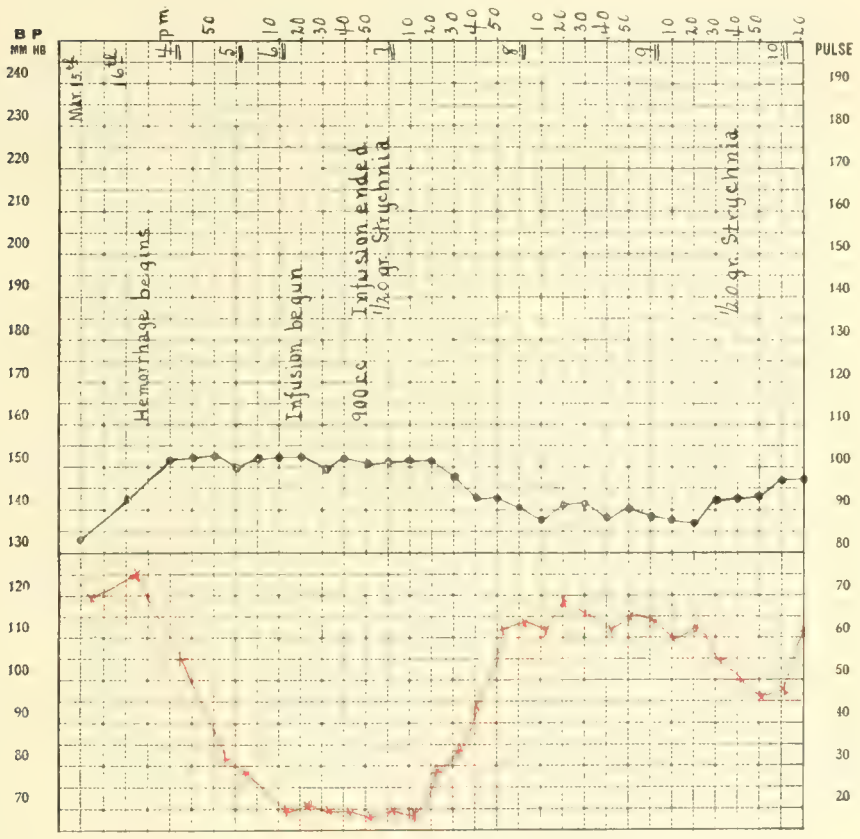
The blood pressure at first was only 60 mm. Hg. Following the administration of one-twentieth of a grain each of digitalin and strychnine, it rose to 70 mm. Hg., and showed no subsequent depression for one hour and a half preceding the beginning of the chart. The chart shows the control obtained over the blood pressure by means of intravenous transfusion of adrenalin solution, with irregular oscillations during the period of adjustment of the strength of the solution—with final success in obtaining a uniform pressure adequate for the continuance of life. The case, after many hours, terminated fatally by sudden, apparently cardiac, collapse. The heavy interrupted line below indicates the period during which the adrenalin was flowing into the vessel.

sive stimulant hypodermic injection, until the physiological level was reached and maintained without further treatment. In the three other cases "improved," the benefit was merely temporary, and after moderate rises in blood pressure (10 to 20 mm. Hg.) lasting from one-half to one and a quarter hours, the pressure again fell to its previous level in spite of stimulation and mechanical measures directed to increasing peripheral resistance. In two cases drug stimulation of the vasomotor center was ineffective, maximal doses of strychnine and digitalin failing to raise the blood pressure to any appreciable extent, or for more than a few minutes. Not one of the doses of any of the three drugs employed was followed by a fall in blood pressure, either after or in the absence of an initial rise, which might fairly be attributed to their action. Thus, in ten cases of shock heavily stimulated by successive doses of strychnine and other central vasomotor excitants, there was no further depression produced by any dose, nor by the summation of many doses, of those drugs. In every respect, as far as the blood pressure response to stimulants and its coincidence with the general symptoms of improvement is concerned, the behavior of these cases was exactly similar to that of patients suffering from toxic or hæmorrhagic hypotension and subjected to the same treatment (Charts VI, VII, VIII, IX).

In three of the cases strictly peripheral vascular stimulation was practised by means of continuous transfusion with adrenalin chloride in salt solution (Crile, p. 249), and it was always possible in this way to raise the blood pressure to any desired level. Central stimulation in these cases was not effective, or produced only transient rises in blood pressure. All three died rather suddenly, apparently from cardiac syncope, during the administration of the adrenalin—though one case was kept alive by this means for fourteen hours after the exitus was considered imminent. In one of these cases, at least, the hypertension due to too rapid preliminary administration of the adrenalin was associated with evident brightening of the patient's mental condition, improvement in the respiration, and apparent temporary relief of the shock. In the other two, this association was not so obvious, and in spite of the normal blood pressures artificially maintained by excessive peripheral vascular constriction, the general nervous loss of tone was not modified. Central stimulants, on the other hand, when they were effectual in raising the blood pressure to a safe level, always produced easily visible signs of improvement in

the general condition, and the measured increase in vascular tone under their influence proved to be a reliable guide to the final prognosis, and not a deceptive one, as where adrenalin was used (Chart IX).

CHART No. X.



Pulmonary Hæmorrhage of 1100 cc.

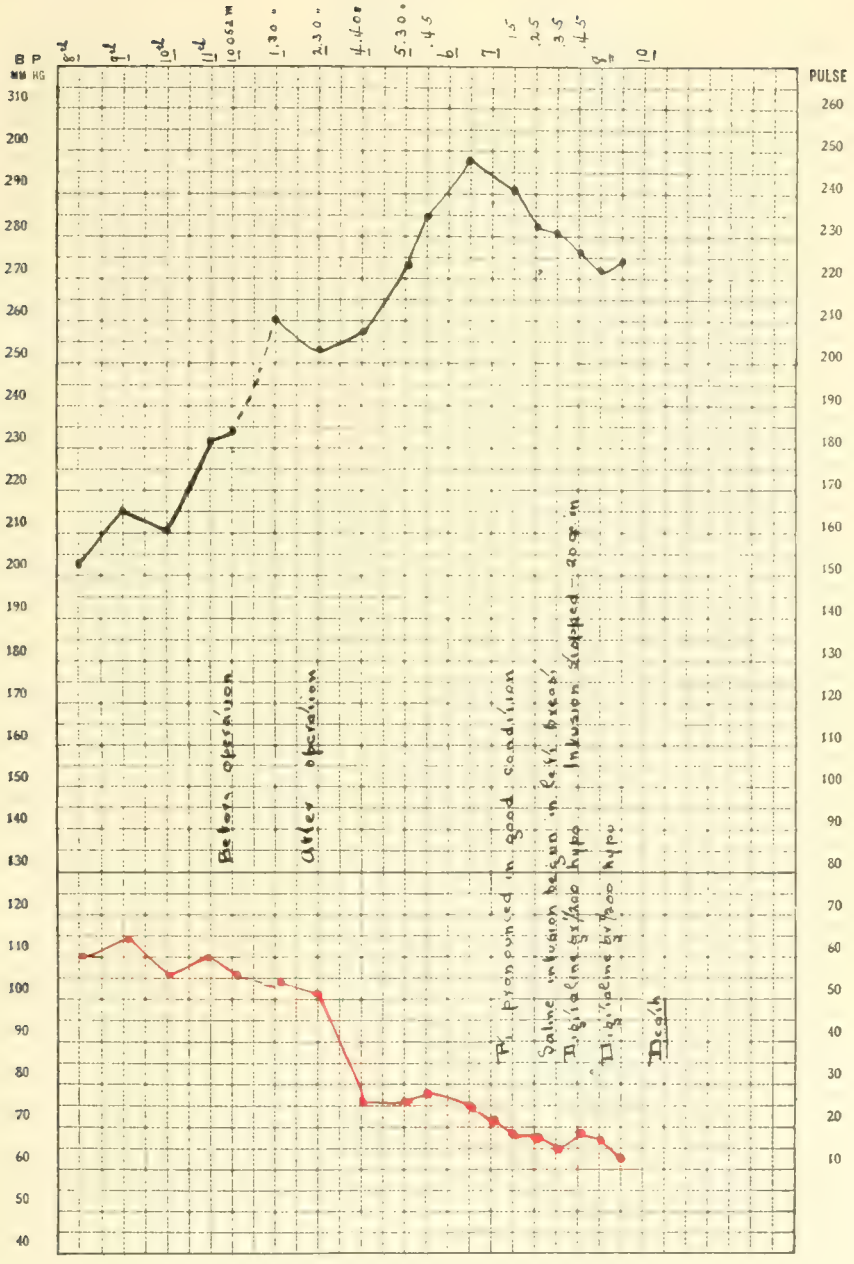
(1) Saline infusion without pressor effect.
(2) Strychnine giving marked pressor response.

Crile (17) has supported, by a series of experiments on the dog, the theory which regards shock, as seen in the clinic, as a manifestation of exhaustion of the vasomotor center by peripheral stimuli. He considers central stimulation by strychnine to be identical in its

effect on the controlling nervous mechanism of the vasomotor system with that of excessive afferent impulses. Hence, he believes the treatment of shock by central stimulants, such as strychnine, to be as irrational as would be treatment by continued peripheral stimuli, and as certain to produce uniformly evil results. He says (op. cit., p. 250): "There is no practical distinction to be made between external stimulation of the vasomotor center, as in injuries and operations, and internal stimulation by vasomotor stimulants, as by strychnine. Each in sufficient amount produces shock, and each, with equal logic, might be used to treat the shock produced by the other. Stimulants of the vasomotor center are contraindicated" in shock. Crile would attempt to obtain complete physiological rest of the center, and believes the therapeutics of shock are justified only in attacking the vasomotor periphery. In collapse, however, stimulants (central) may be useful because the centers are not exhausted.

This theory of the intimate nature of shock, the experimental basis for it, and its therapeutic corollaries, are so convincing in the detail of their elaboration that any criticism of Crile's conclusions by purely clinical observers, on the basis of a limited material, must be very tentative. It is possible to reconcile these views with our results by assuming that all cases of post-traumatic vasomotor depression, transient or of long duration, which react favorably to internal stimulants, must be excluded by that fact from the category of shock. Under such conditions, five of our ten cases certainly, and three more probably, were not in shock at all, but were merely suffering from vasomotor loss of control of an indefinite nature. Such a readjustment of terms to facts—for the question in its final form becomes merely one of nomenclature—does not appear necessary or justifiable. In the severity of the initial trauma, the degree of the hypotension of the pulse, the character of the blood pressure reaction (further depression) after unavoidable peripheral irritation, and the duration of the unfavorable symptoms, all the cases observed conformed absolutely to the well-recognized picture of true shock as it is seen in the clinic. In none of the cases did the blood pressure show any tendency to return to the normal spontaneously, or in the absence of active treatment. Considering the degree of shock present alone, and without regard to the necessarily fatal character of the injuries in themselves in one or two of the cases, it was impossible to say at any time before treatment was begun, that the patients who eventually recov-

CHART No. XI.



Fatal Post-Operative Shock.

Subsequent to laparotomy and laminectomy in a child two months old. The profound character of the depression was indicated three hours before the palpable pulse character or the general symptoms gave any warning, by the course of the blood pressure curve. The rising pulse rate was thought to be of little significance in so young a subject. One hour before death, disregard of the blood pressure led to the giving of an absolutely favorable immediate prognosis.

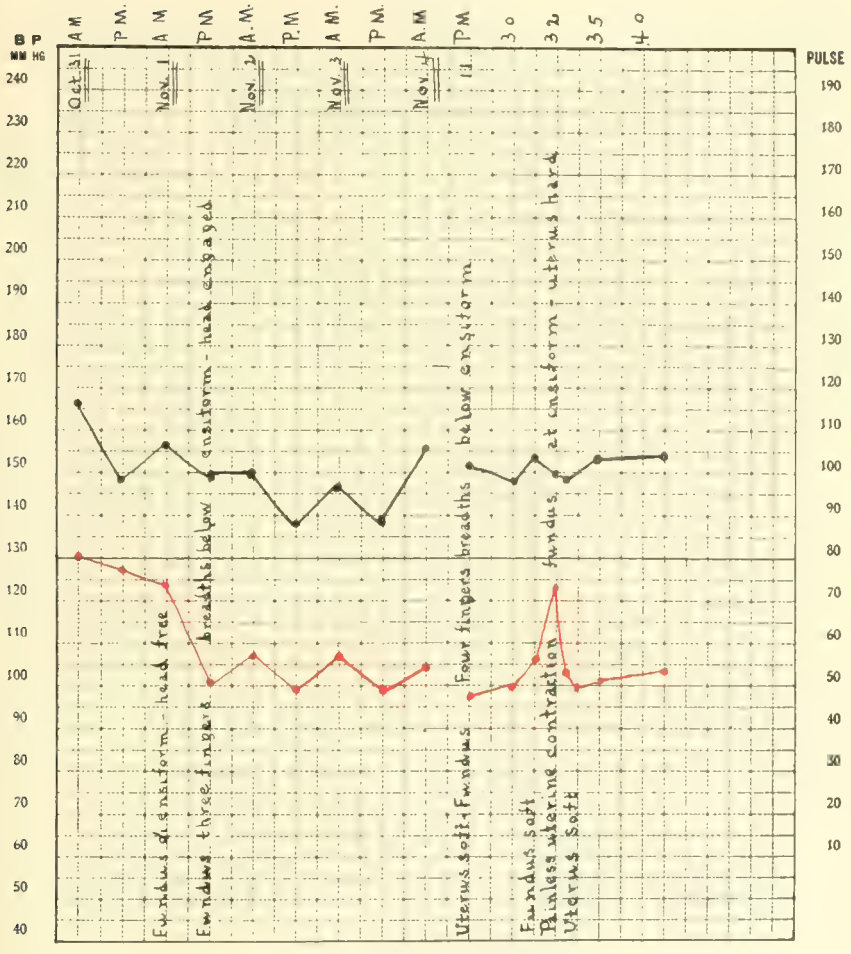
ered after stimulation were any less severely shocked than those who died. In fact, the one case whose condition was, when first seen, apparently more hopeless than that of any of the others, was one whose reaction to stimulation was the most brilliant of all, and whose complete recovery was most certainly obtained by this means.

In two cases (20 per cent of the whole number stimulated) there was no response on the part of the vasomotor center, as determined by blood pressure measurements, to large doses of strychnine and digitalin. Stimulants here certainly did no good, and it is possible that their final effect may have been harmful, though there was nothing in the course of the blood pressures after stimulation to support such a view. We have shown elsewhere in this paper that in the toxæmia of typhoid fever, the condition above all others where the good effects of stimulation can be most exactly judged from its effect on the blood pressure, a similar absence of reaction is also sometimes met with. It is one of the most certain signs of the approach of death in this disease that strychnine and digitalin should fail to produce a rise in blood pressure. Yet there is no question that deeply toxic typhoid fever patients should be freely stimulated in every instance, it being impossible to determine beforehand whether stimulants will be effectual or not. Similarly, we believe that all cases of shock, or of post-traumatic vasomotor depression, should be subjected to free central chemical stimulation. Some of these cases, we believe the majority, will be definitely benefited by such treatment. It is equally certain that others will not be helped in any way—but until we can separate these cases from the more favorable ones by tests other than therapeutic, it does not appear that they should be refused what may be their only opportunity of final recovery.

4. Hæmorrhage.

With the present degree of perfection in the technique of hæmostasis on the operating table, and the appreciation of the predisposition created in the nervous centers by active hæmorrhage to the shock-producing factors, it is comparatively rare to have any very great loss of blood occur as the complication of the majority of surgical procedures. Even the wide denudation of the chest wall accomplished in the complete operation for mammary carcinoma is performed to-day without significant hæmorrhage. In some cases, however, loss of a certain amount of blood is a necessary complication of operation, its

CHART No. XII.



Ninth Month of Pregnancy.

(1) Fall in blood pressure with descent of the fœtal head into the pelvis.

(2) Uterine contraction (Braxton-Hicks) accompanied by a sharp rise of blood pressure, not associated with sensations of pain or contraction of the muscles of the abdominal wall.

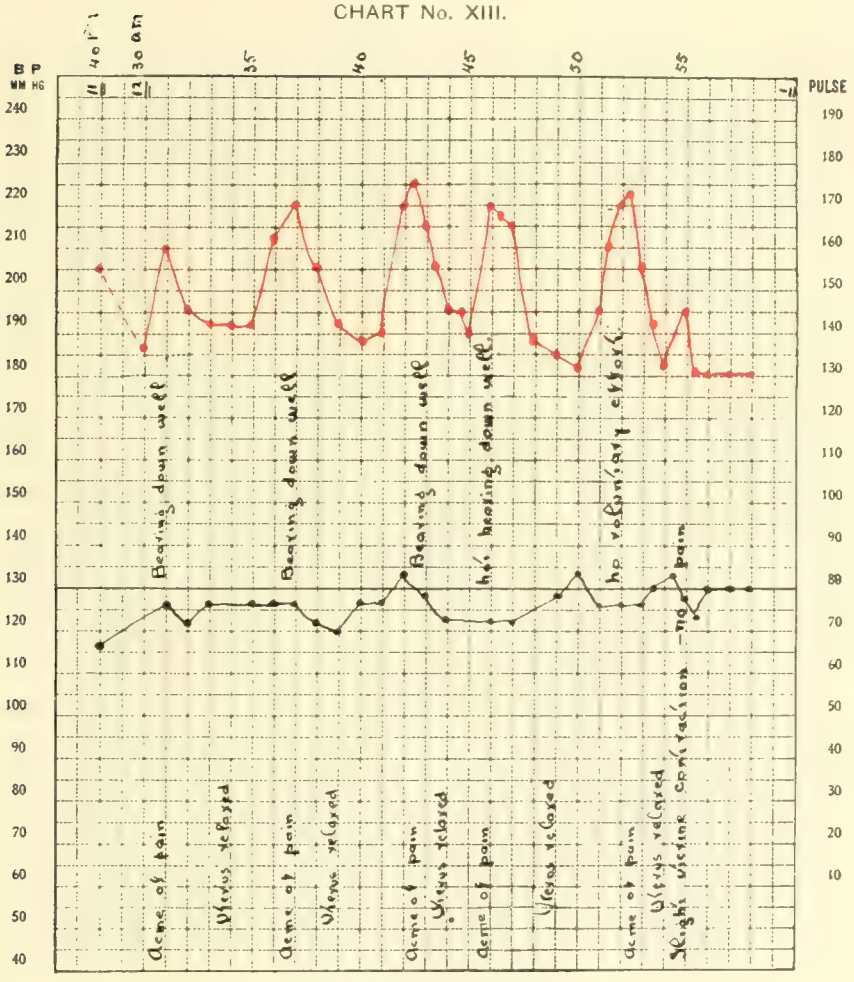
effects on the patient varying with the presence or otherwise of concomitant depressive factors, and largely with the previous general condition of the patient. These effects cannot be more correctly gauged, both at the time of operation and afterwards, than by frequent blood pressure estimations (Charts X, XXIII, XX). As we have shown elsewhere, the blood pressure reaction in hæmorrhage is very characteristic—the charted observations showing a V-shaped curve, whose descending limb marks the continuance of bleeding, while the ascending arm indicates its cessation and the physiological return of the vasomotor system to its normal state. As the formed elements of the blood return quickly to their normal numbers after the depleting effects of a moderately large single hæmorrhage, so the pulse tension in the same case makes typically a quick recovery. Any prolongation of the descending phase, or postponement of the return, after local hæmostasis is complete, indicates that the depressive effect of the loss of blood has been out of proportion to its actual amount, or that a reflex nervous depressive action is also taking place (shock). In any instance of this nature where the shock element can be eliminated or neglected, the measured fall of blood pressure and its duration gives a more exact determination of the severity of the hæmorrhage than does the mere volume of blood lost. Where the local control of hæmorrhage has been uncertain, a continued fall in blood pressure is a sure indication of the ineffectiveness of the hæmostasis.

Perhaps no class of cases responds more actively to central stimulation than those of post-hæmorrhagic hypotension—it being not infrequent to find rapid jumps in blood pressure of 20 to 40 mm. Hg. after moderate doses of strychnia, breaking the otherwise slow and regular ascent of the pressure from its low level. This is especially noticeable in patients on the operating table, who if properly stimulated may suffer but very brief depression after brisk hæmorrhages. This typically rapid rise justifies the caution that stimulants should be applied very sparingly, if at all, in this form of hypotension, until all bleeding vessels have been controlled, in order to avoid the dangers of renewed bleeding upon the establishment of increased intravascular pressure.

5. Post-Operative Conditions.

The value of accurate measurements of arterial tension is shown very strikingly in the diagnosis and treatment of post-operative cardio-

CHART No. XIII.



First Stage of Labor.

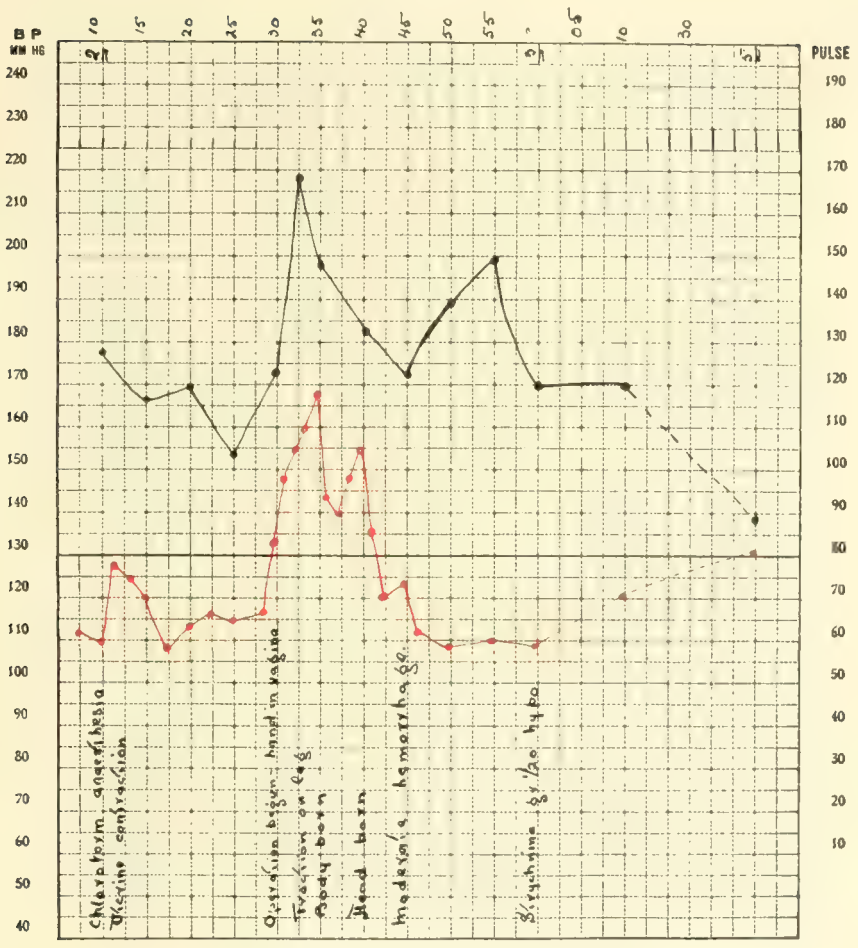
Regular succession of uterine contractions, each one accompanied by a sharp rise in blood pressure, and not proportionate to the amount of voluntary effort expended.

vascular complications. In no class of cases are there found more deceptive variations, or the want of them, in the mere pulse rate—while variations in blood pressure of significant degree are by far more constant. In many patients after operation there is for a time a dread of the onset of post-operative shock or of the occurrence of concealed hæmorrhage, and pulse tension here is of the greatest importance in diagnosis (Chart XI). We have seen shock develop after a major operation to such a degree that the blood pressure fell from 140 mm. Hg. to 75 mm. Hg., while the pulse rate remained below 100 to the minute—in another case hæmorrhage occurred until 1100 ccm. of blood were lost, with marked hypotension of the pulse, before the rate had risen to 90. On the other hand, post-operative tachycardia of high degree may co-exist with a normal or increased pulse tension, when of course the possibility of shock or hæmorrhage is at once excluded, and the search for some other condition as the cause of the symptoms is suggested. In four such cases recently observed, the heart in all beating at about 180 to the minute, and the possibility of shock or concealed hæmorrhage being very great in all, the presence of blood pressures of above 150 mm. Hg. suggested the diagnosis of acute cardiac dilatation, which was confirmed in every instance. It may readily be seen how much our indications for treatment may be modified by early and proper recognition and differentiation of these varied complications. Another and important signal may be early and steadily increasing hypertension, a very constant warning of the onset of post-operative renal insufficiency, in which active measures for increasing elimination may thus be justified long before attention would otherwise be called to their advisability.

IV. Blood Pressure Observations of Obstetrical Interest.

(1). *Pregnancy.*—We have made no systematic observations on the intravascular tension in the earlier months of pregnancy, but from casual measurements recorded, it seems unlikely that there is any very marked deviation from conditions as seen in non-pregnant women. There has been no attempt made to determine whether the menstrual hypertension manifests itself during the physiologic suppression of the periodic discharge. It is interesting that the painless uterine contractions of Braxton-Hicks, as seen in the months before term, are accompanied by blood pressure changes quite analogous to those occurring in connection with the later true labor pains, and differing from them only in degree. (Chart XII.)

CHART No. XIV.



Podalic Version: Contracted Pelvis.

(1) Low tension under chloroform anæsthesia.

(2) Rise in blood pressure with uterine contraction.

(3) Pelvic reflex rise in blood pressure on introduction of operator's hand into the vagina, exaggerated during traction on the child.

(4) Fall in blood pressure with delivery of the child.

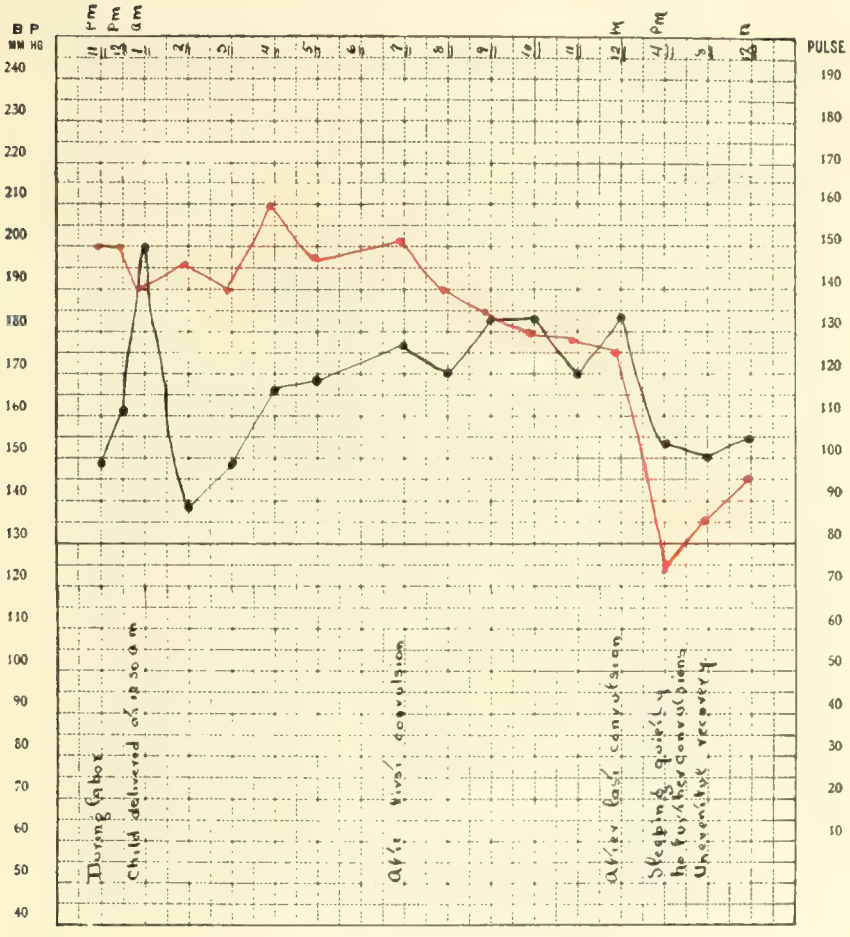
(5) Rise in blood pressure post partum following the administration of strychnine.

That the presence of the growing fœtus within the abdomen, and the local vascular alterations accompanying pregnancy, should in some way modify the ordinary blood pressure conditions is to be expected when the relation of the great splanchnic reservoir to the peripheral blood supply is considered. It is probable that the potential effects of the altered uterine circulation on the general systemic blood pressure are either neutralized or altogether masked by the tendency of the enormously enlarged uterus to act in the manner of abdominal compression mechanically applied, by raising and keeping the blood pressure at a level more constant than would otherwise be the case. That the mere bulk of the fœtus is thus mechanically a factor in maintaining the relatively normal state of tension in the peripheral vessels during pregnancy, is evident from the fact that the engagement of the fœtal head in the pelvis in the ninth month, with its consequent reduction in the total intra-abdominal fœtal mass, is always accompanied by a fall in blood pressure from the previous level. (Chart XII.) The blood pressure then continues at this lower level until the actual onset of labor, or some complication, again alters the vasomotor equilibrium.

(2). *Labor.*—The onset of labor is normally accompanied by an increase in the mean systolic blood pressure. This rise is mainly due to the marked hypertension attending the forcible uterine contractions, and each labor pain is accompanied by a rise in blood pressure, continuing through its course, and exactly parallel with the state of contraction of the uterine wall. (Chart XIII.) During the interval of the pains the blood pressure is somewhat above its previous level, owing in part to the effects of excitement. That the rise in blood pressure accompanying the pains is not wholly or even in large part due to contraction of the abdominal walls or of the muscles in general is evident from its constancy when these pressor factors are not present. Even when the patient is well under the narcotic influence of chloroform and the general musculature and the abdominal parietes are completely lax, each uterine contraction manifests itself by a blood pressure rise which is in general almost as great as when the patient is fully conscious and abdominal contractions are efficient. (Chart XIV.) With the patient in a state of exhaustion, the blood pressure is low during the pain intervals, but rises with each pain, irrespective of the amount of voluntary effort put forth by the patient.

As labor progresses and the pains become more frequent and more

CHART No. XV.



Puerperal Eclampsia.

(1) Hypertension during labor.

(2) No relief of the abnormally high blood pressure following delivery—only a very temporary fall occurring.

(3) With the persistence of hypertension, development of eclamptic symptoms and convulsions seven and a half hours post partum.

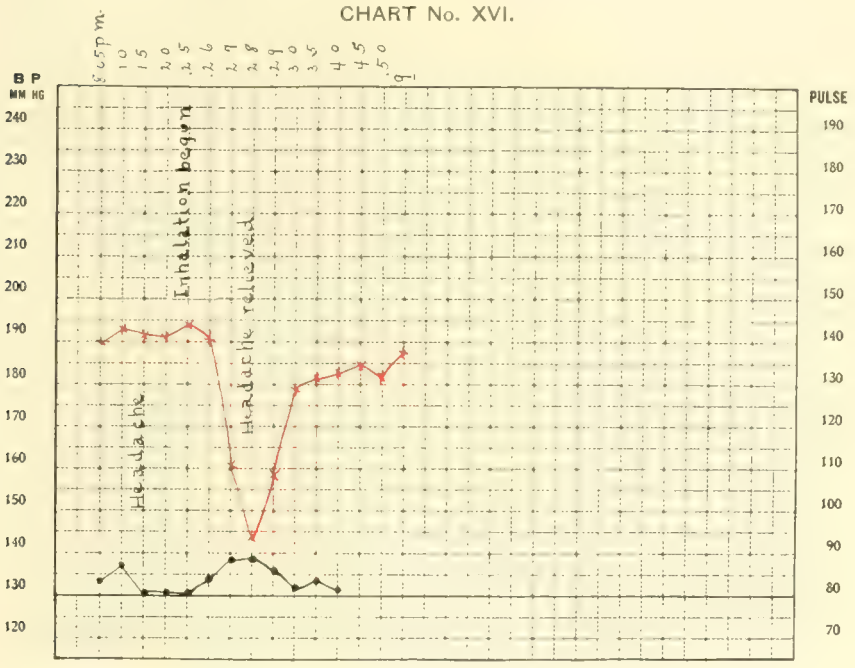
(4) With the relief of hypertension (by the usual methods) disappearance of the eclamptic features.

vigorous, these features are more pronounced. At the moment of expulsion of the child there is a final and distinct fall in systolic blood pressure to a level somewhat below that normal for the individual, but not greatly so unless there is much exhaustion or hæmorrhage is excessive. If this complication occurs, the fall in pressure is progressive, and as in all conditions where there is much loss of blood, the persistence and degree of the depression is a measure of the severity of the hæmorrhage in its effect on the patient. During the passage of the child through the pelvis the blood pressure rises to a degree even greater than during the pains of the first stage of labor. If the delivery be instrumental, or if in the case of a podalic version very active traction is necessary to end the labor, an extreme degree of hypertension is produced. This "pelvic reflex" rise in blood pressure occurs, even under anæsthesia, when the whole hand is introduced into the vagina, but is most marked when traction is made on the descending child, and great pressure is thus produced on the walls of the pelvic canal. (Chart XIV.) The blood pressure curve of an instrumental delivery per vaginam resembles closely that obtained during rectal operations, with forcible dilatation of the sphincter ani.

(3). *Eclampsia.*—This complication is of special interest in view of the uncertainty that prevails as to its etiology. Evidently a toxæmia, with many points of clinical resemblance to uræmia, it is noteworthy, like the latter condition, in having hypertensive effects on the systemic blood pressure. In the parallelism of the hypertension with its general symptoms and in the disappearance of this hypertension with relief of the symptoms, it differs in no way in its effect on the pulse from the ordinary uræmia of nephritis, though this indicates no necessary identity of causation for the two conditions, of course. But it is especially with regard to the early recognition of the onset of eclamptic features in any case, and the possibility of instituting prompt and vigorous treatment for their relief, that systematic blood pressure records may be of value to the obstetrician.

The onset of hypertension of the pulse at any period in pregnancy should always excite the apprehension of eclampsia, in the absence of other recognized causes of abnormally high blood pressures. If albumin be present in the urine during pregnancy, there is practically certain to be some degree of hypertension, but it would seem that the persistence and increase of this pathological state of pulse tension is much more directly indicative of approaching eclampsia, and of

the actual existence of a minor degree of intoxication, than is any change in the urine. Patients with eclamptic features have blood pressures very much above normal during labor, both during the pains and in their interval. If this hypertension persists in the third stage, if the physiological fall attending delivery be absent, or slight in amount, measures tending to the relief of the toxæmia are almost as urgently indicated as if convulsions had already occurred.



Effect of Inhaling a Bead of Amyl Nitrite.

In the cases observed, this post-partum hypertension has always been associated with subsequent convulsive attacks. (Chart XV.)

V. Blood Pressure Observations in Medical Conditions.

(1). *Hypertension.*—A pathologically heightened blood pressure is an essential feature, as has long been recognized, in the symptom complex of a large group of cases seen more especially in medical practice. Often its existence, as in cases of coma of obscure nature,

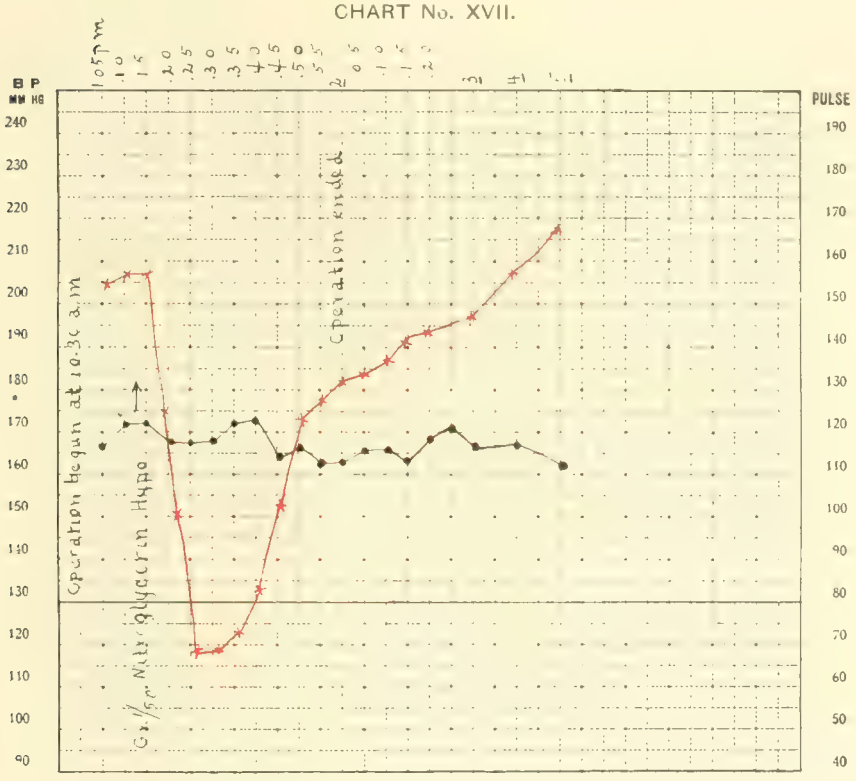
may be when determined an important aid in diagnosis. (Chart XX.) As regards prognosis, its degree is one of the ready measures of the damage done by an existent nephritis, chronic toxæmia, or valvular lesion, and in treatment, the indication it affords is as precise on the first examination, as its reduction is important as an index of the success of the methods employed.

Practically all patients presenting themselves for treatment for acute or chronic nephritis, with or without uræmic symptoms in varying degree, for the chronic toxæmia of gout and lead-poisoning, and for all the effects of general arterio-sclerosis, aortic aneurism, valvular and muscular heart lesions, short of loss of compensation, present the picture of hypertension. The measured blood pressure in these patients will vary from 160 to 250 mm. Hg., or exceptionally even higher, depending on the age and general condition of the patient, and on the accessory features of the case. In all, the reduction of the tension of the pulse, whether directly by the use of vascular dilatant drugs, or indirectly by promoting the elimination of toxic materials, is always an important and often the one vital indication in the handling of the case. The fact that is not generally understood, and the one we wish especially to emphasize, is that the course of the changes in blood pressure in these patients, as measured from hour to hour, or day to day, is almost precisely parallel with the improvement in the patient's fundamental morbid condition. No touch, however delicate, can appreciate the difference of ten per cent in the height of the blood pressure that may be effected, let us say, by a night's rest in bed, or by a course of purgatives and diaphoretics, in uræmia. But that ten per cent of change towards the normal is an absolute sign of an improvement in its early stage, which otherwise would not be appreciated until much further developed. Its determination with exactness by the Riva-Rocci instrument is so readily made as to make a knowledge of it essential to good practice.

The indication for a blood pressure lowering treatment once given, its trend and duration can be exactly controlled only by means of accurate blood pressure measurements. The transiency of the effects of moderate venesection, or of nitroglycerin after the first forty-eight hours—the much greater permanence of the reduction following free purgation, sweating, or the more stable nitrites—can all be gauged and provided for, and that measure can be immediately adopted which will best relieve the urgency of the individual case.

It is not our purpose to give here a detailed report of studies in the reduction of hypertension of the pulse—the field is too large for the limits of this article. We wish merely to call attention to the exactness that is now possible in the observation of a symptom, which,

CHART No. XVII.



Effect of Nitroglycerin Administered during Operation to Relieve Hypertension.

Pressure before and during operation had ranged between 200 and 260 mm. Hg.

while its importance is a matter of common acknowledgment, has been susceptible in the absence of an efficient instrument, of only the most loose and inaccurate evaluation. (Charts XVI, XVII, XVIII).

Under certain conditions, hypertension has a very different significance from that discussed above, and far from being an indication for venesection as the most promptly acting corrective, calls rather

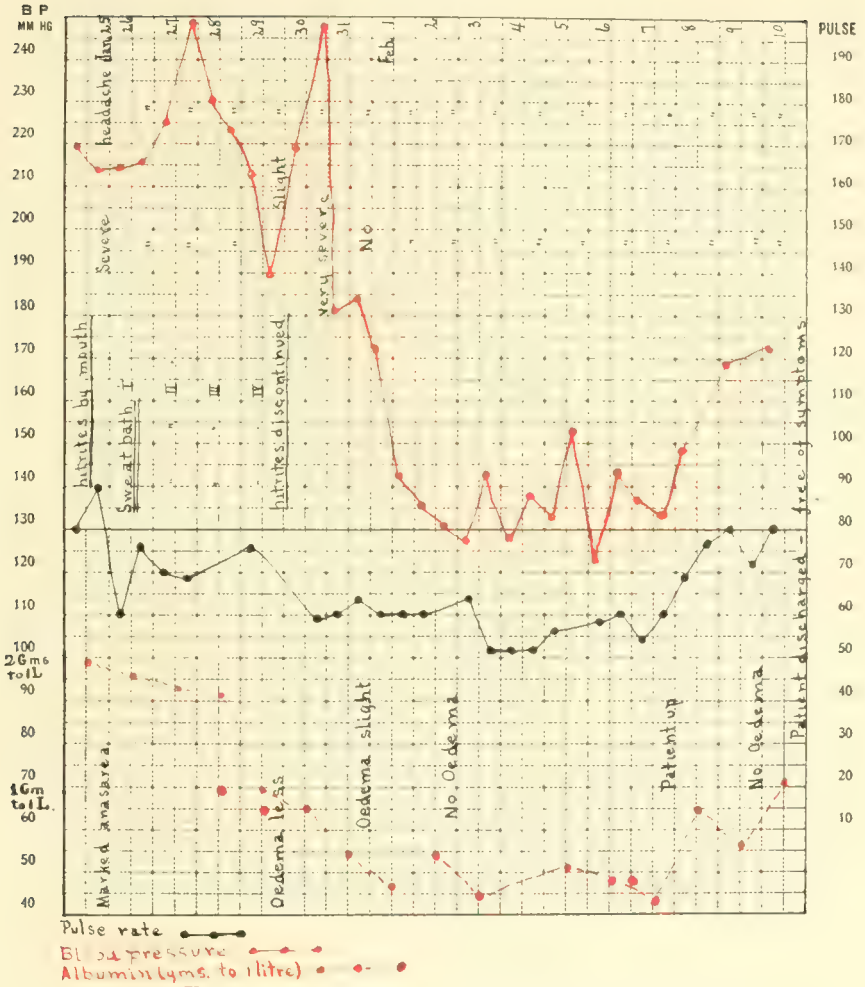
for support until surgical relief can be obtained, (Charts VIII, XIX, XX.) We refer to the cases of rapidly increasing intracranial tension, when the vicious circle described by Cushing (18) has been set up, and the steadily mounting blood pressure is the sign of the struggle being made by the peripheral vessels against fatal anæmization of the medulla by local pressure. In these cases the highest blood pressure readings yet reported have been obtained—approaching 400 mm. Hg. in one instance observed by us—and this extreme degree of hypertension constitutes one of the most valuable points of diagnosis in otherwise obscure cases of intracranial hæmorrhage. The treatment, as Cushing has discussed it, is not medical, but the recognition of the condition before autopsy in a certain number of cases of coma of clinically obscure nature, may rest entirely in the hands of the man who can make exact blood pressure measurements.

Gradual increase of intracranial tension, on the other hand, seems to have no effect in raising the general arterial tension. In patients with brain tumor of some years' duration, with persistent, long-continued headache and optic neuritis of high degree, we have never observed any increase of blood pressure. In some cases it has been below normal, in none higher than was appropriate for the degree of arterio-sclerosis sometimes present, and in none has there been any constant association between variations of the severity of the headache and variations in blood pressure.

Hypertension in Tobacco Smokers.—A rise in blood pressure produced by alcohol is frequently assumed to be one of the factors associated with a toxic deterioration of the vessel wall, in the production of the arterio-sclerosis consequent to long-continued tippling. We have shown that alcohol *per se* is incapable of raising the blood pressure, and any hypertension associated with its use must occur at a period when toxic damage to the excreting organs has impaired their eliminative powers. In other words, if habitually high blood pressures are an efficient factor in the determination of vascular fibrosis, they are not active early in the career of the alcoholic, and only become so when a toxic or other sclerosis of the renal vessels has been established. Alcohol does produce hypertension, but only secondarily by affecting the nutrition of organs, the disordering of whose functions from any cause is associated with heightened pulse tension.

But there is another form of voluntary intoxication, that enjoyed by the tobacco smoker, which has a decided influence on the arterial

CHART No. XVIII.



Uræmia in Chronic Parenchymatous Nephritis.

(1) On admission, hypertension, with anasarca, headache, mental hebetude, and marked albuminuria.

(2) Fall in blood pressure under depressant (nitrites) and eliminative (sweating, purging, diuresis) treatment.

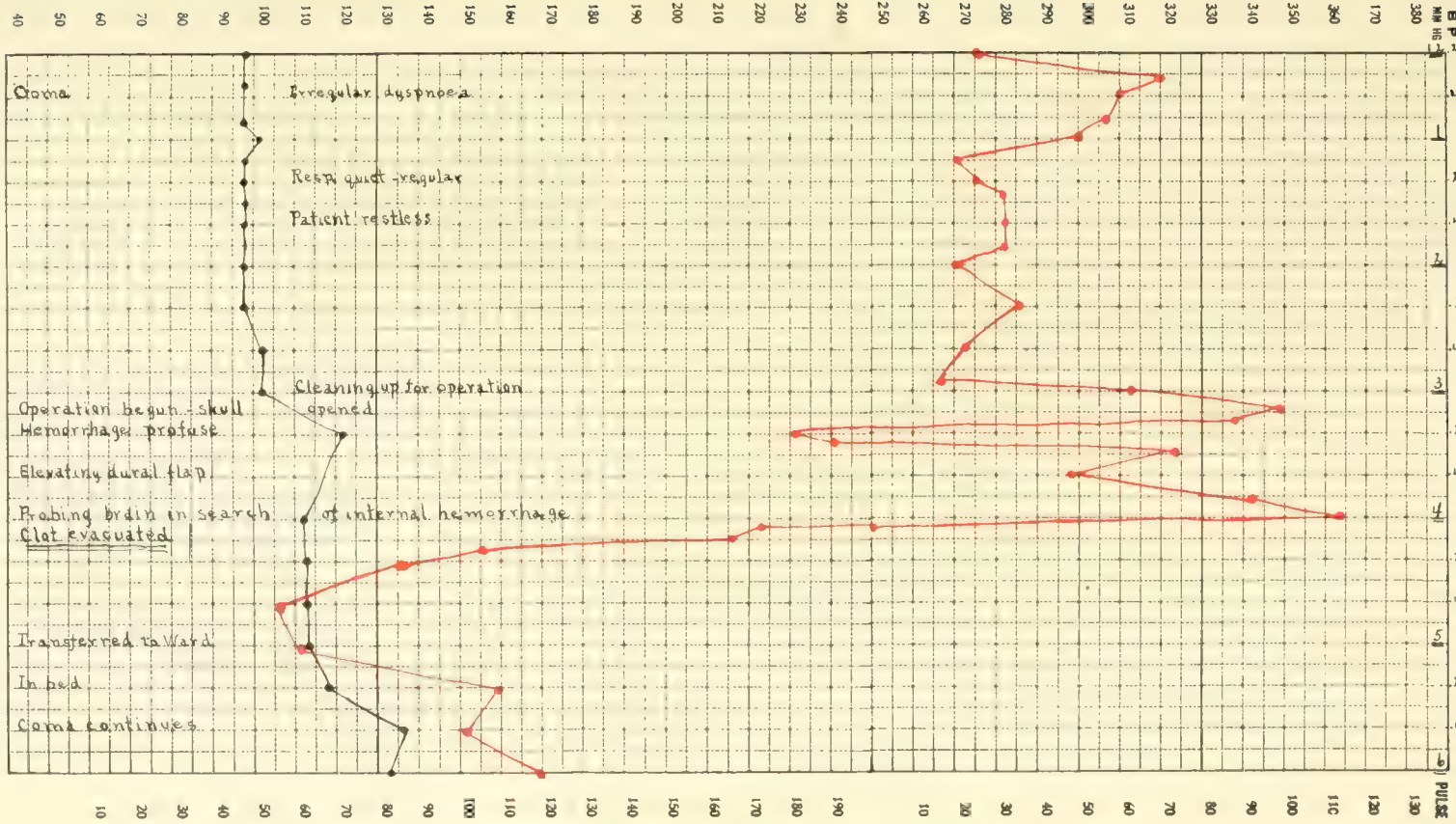
(3) Improvement in all symptoms parallel with the diminution of blood pressure, and retarded during transient return of hypertension.

(4) Parallelism of curves of blood pressure and albumin per cent, with rise in both when patient is allowed to get up.

(5) Transient effect of depressant drugs on both pulse tension and general symptoms, contrasted with the more lasting effects of active elimination through the skin, bowels, and kidneys.

blood pressure. (Chart XXI.) There are occasional references in European literature to a tobacco sclerosis of the vessels, but without any satisfactory explanation of the mode of its development, and the study of smokers' blood pressures promised to throw some light on the subject. It has been found that the inhalation of tobacco smoke, in those long addicted to the habit as well as in the neophyte, is always attended by a very considerable increase of pulse tension, and this in spite of the mentally and nervously sedative effects of the practice. Smoking without inhalation of the products of combustion has much less effect, and in many cases none; and the whole tendency of the tobacco smoker's blood pressure curve shows that its rise is due to the action of some toxic bodies after absorption, and not to any peripheral reflex from irritation of the respiratory mucous membranes. The rise in blood pressure is gradual, increases regularly until a rise of 10 to 30 mm. Hg. has been established, and is greatest when "strong" tobacco is used and at the time when the smoker feels a sensation of definite intoxication. It is most marked when the smoke of a heavy cigar is inhaled, almost as great with an old pipe, and least when the tobacco consumed in the form of a cigarette. This tobacco hypertension is not merely transient, but endures for from one to two hours or even longer after the smoke is finished—long enough to make it certain that a regular smoker maintains by his habit an abnormally high blood pressure during all of his waking hours. Uncertain as it is just how great a part undue vascular strain when long continued can play in the production of arterial degeneration, in the absence of all other factors, it must have some influence, if only a slight one, in lessening the resistance of the media to circulating toxic substances, and we have in tobacco smokers a combined toxic and hypertensive condition. It may be that the toxic element is the overwhelmingly important one in the arterio-sclerotic etiology, and that alcohol, while not primarily a hypertensive poison, is a much more injurious one as regards the nutrition of the vessel walls than that given off by burning tobacco. General arterio-sclerosis and tobacco smoking are both so widespread among the adult males of the civilized world, that a statistical study of their relation would probably give very unsatisfactory and contradictory results. However, in view of the facts presented, and of the fragmentary state of our knowledge as to the intimate etiology of sclerosis in general, the tobacco habit appears worthy of inclusion with the other chronic intoxications in the list of possible or probable causative factors.

CHART No. XIX.

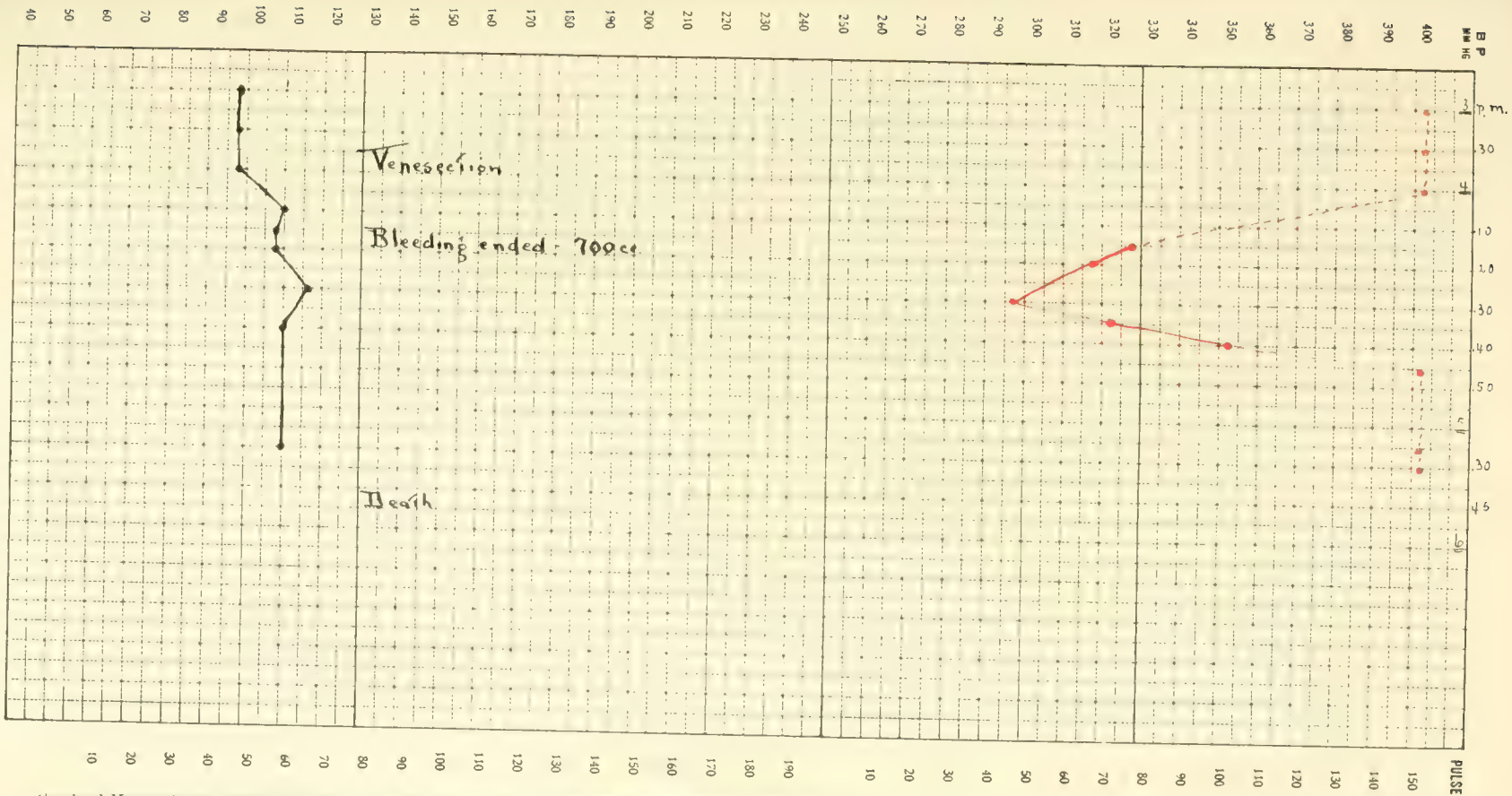


Intracranial Hemorrhage (Apoplexy) with Exploration of the Brain and Evacuation of a Large Clot.—(1) Persistent extreme hypertension with slow (vagus) pulse, resulting from mechanically increased pressure on the medulla. (2) Exertion (preparation for operation) increasing the blood pressure by exciting further hemorrhage, and so increasing intracranial tension. (3) Elevation of bone flap, and brisk external hemorrhage, reducing the blood pressure momentarily. (4) Immediate complete relief of the hypertension on evacuation of the compressing intracerebral clot, with further depression due to slight shock, and subsequent return of blood pressure to the normal level (arterio-sclerotic patient).

(1). *Hypotension.*—Abnormal lowering of the blood pressure, except in the extreme degree that is practically only met with in states of shock, is not in itself necessarily and immediately harmful, and it is of interest rather as a sign of general or cardio-vascular weakness, than as a condition in itself whose relief is urgently demanded. In a patient with a "weak and fluttering" pulse, as it is generally described, and a measured peripheral blood pressure constantly below 60 to 80 mm. Hg., there is certainly a dangerous impairment in the nutrition of all the tissues, especially those whose activity is immediately necessary to the continuance of life, and which depends directly on the reduction in the amount of blood which can be furnished to the tissues at the lowered rate of supply. A low and stationary blood pressure, however, does not necessarily demand relief as does the continuously high pressure—considering only hypotension as seen by the internist, it rarely in itself excites alarm, except as it gives information of already existing and possibly increasing weakness in the musculature of the heart and vessels. Hypertension, in other words, may be regarded both as a symptom and as a complication, a part of a morbid process, and in itself productive of further morbid changes—while hypotension, clinically considered, is always a symptom, a result and not a cause, except when it is so extreme and permanent as to constitute shock. Then it becomes in itself a destructive factor, and its removal a matter of vital import, entirely apart from the disputed manner of its production.

In the medical wards low blood pressures are found in the cachectic states and in all conditions associated with impairment of the general nutrition, in all acute toxæmias towards the end and when profound, and in some intoxications as an early and constant symptom. In the anæmias, whether hæmorrhagic or toxic, acute or chronic, we have been unable to substantiate the view frequently expressed that they are associated constantly with abnormally high blood pressures. We have rather found a uniform tendency to hypotension in all these cases, frequently modified, to be sure, by co-existent arterial disease or other hypertensive influences, and only very roughly proportionate to the degree of anæmia present. In progressive anæmias, it is true, the blood pressure falls generally with the fall in the actual blood count, but not parallel with it by any means, and apparently entirely as an expression of the increasing general asthenia. The high, bounding pulse, empty between the beats, of the profoundly anæmic

CHART No. XX.



Cerebral Hemorrhage (Apoplexy) with Coma. (1) Extraordinary degree of hypertension probably only producible as an effect of increased intracranial tension. At autopsy a coagulum was found projecting into and completely filling the left lateral ventricle. (2) Transient lowering of blood pressure produced by venesection—undertaken in view of the erroneous clinical diagnosis of uræmic coma. Only during the bleeding could the blood pressure be accurately determined, the available manometer only registering to 350 mm. The maximum blood pressure was far above this, probably considerably above the estimated height of 400 mm. of mercury. No observations were taken during the period of vasomotor collapse which precedes death in these cases. See Chart No. VIII.

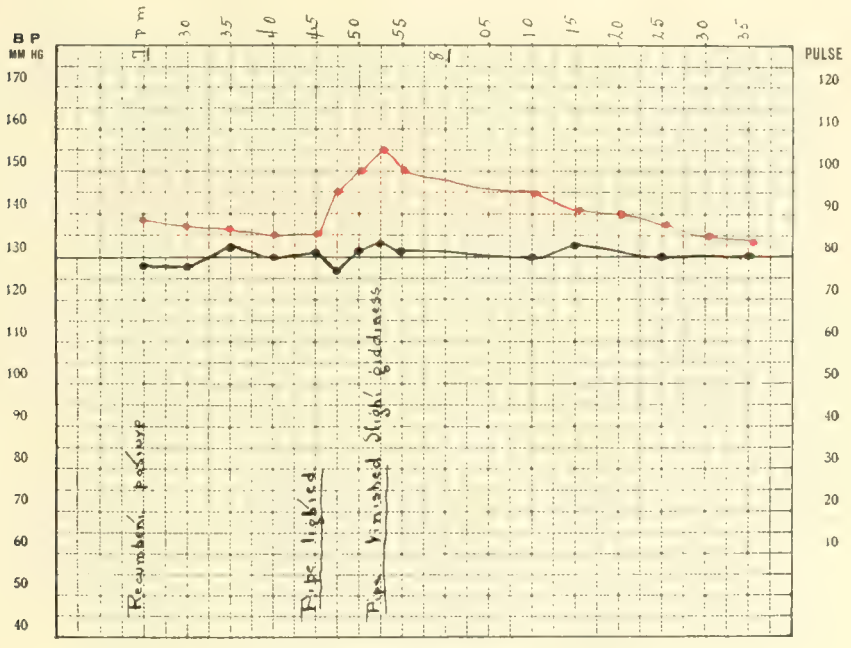
patient, gives an impression to the finger of heightened tension during systole, but actual experiment will show that much less pressure than normal applied over the brachial artery will suffice to obliterate pulsations at the wrist.

A fall in blood pressure, more or less rapid in varying circumstances, is of course the constant precursor of death in all cases. Practically immediate in development in the sudden death of aortic disease, its onset is in most instances much more gradual, and its progress is only one feature in the march of the process of dissolution. In any case where the modification of unduly low pressure is indicated, it is the progressive fall, no matter from what level, that gives the indication, and not the figure expressing the measured force of the pulse at any stated moment. The attempt should be made primarily to interrupt the progressive fall in tension, to maintain the level already present. If that is not possible, it is certain that the patient's vitality is not adequate to maintaining a higher level of blood pressure artificially produced by medication, and that death is imminent. A falling blood pressure, even if it be not at the moment below the normal limits of variation, which cannot be relieved or interrupted in its course by appropriate stimulation, we have found to be a most constant and reliable signal of the approaching exitus. When the pulse tension is low, even more than when it is high, it may be said of systematically charted blood pressure observations that it is the curve of their direction from which the most valuable information can be derived, rather than the position of that curve in relation to the normal level. It is impossible, even within wide limits, to name a degree of measured hypotension which is in itself of immediately or remotely dangerous significance. A patient with a blood pressure of 60 mm. Hg. is *probably* in very serious condition, but more cannot be said without a fuller knowledge of the other features of the case, and especially in the absence of any information as to whether the blood pressure is rising or falling at the time of observation. But bearing in mind the fact that continued vasomotor depression, as expressed by a falling blood pressure, is the first and most striking effect of general vital exhaustion, it is possible to study each case for itself, and to gauge the importance of any given low pressure at the bedside by its relation with other symptoms.

There is a false apparent hypotension, due to what we find to be a very common defect in the faculty of touch, which is frequently

considered to be real and significant in patients whose pulse has been examined by the finger alone. If the pulse be small, that is, if the difference between systolic and diastolic pressures be unusually little, the palpation judgment of tension, made by however skilled an observer, is practically sure to fall short of the actual state of the blood

CHART No. XXI.



Tobacco Smoking.

Effect of inhaling the smoke from an old and "heavy" pipe.

pressure. Especially is this true if the heart's action be rapid, as the mental association between the ideas of "weakness" and rapidity of the pulse is a close one. Such a false diagnosis of hypotension is most frequently made in cardiac diseases, especially where there is more or less irregularity of the pulse, and must in general be an error often indulged in, since constriction of the peripheral arteries tends always to reduce the size of the pulse wave, while raising the actual tension.

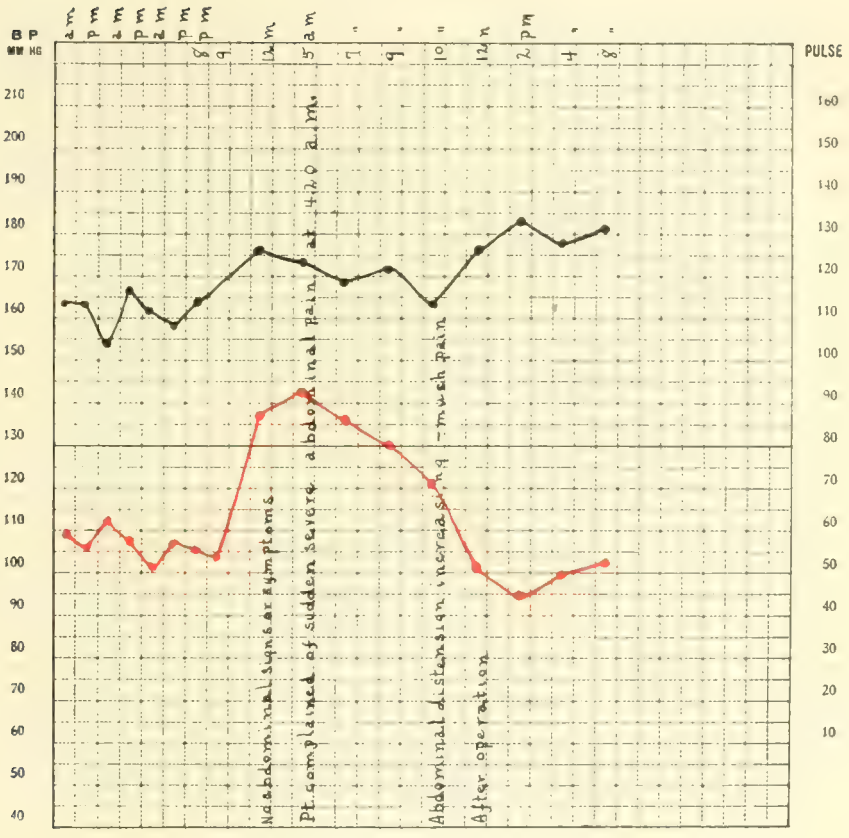
The Blood Pressure in the Acute Infections.—Any acute intoxica-

tion with bacterial toxins, if severe, tends finally to vasomotor paresis, and hence to the production of low pulse tension. On the other hand, where the intoxication is not unusually profound, but corresponds to the type or average as commonly seen in practice, the acute infectious diseases vary considerably in their effect on blood pressure. At the one extreme may be placed typhoid fever, where hypotension, progressive even up to the period of convalescence, is the characteristic vasomotor sign. At the other is acute lobar pneumonia, as seen in previously healthy adults, in whom a moderate or even high degree of hypertension is almost as typical of the ordinary case. Intermediate between these two is the whole group of infections of common occurrence in this latitude, with no characteristic curve of blood pressure, aside from the reduction common to all in the later periods, of no ominous meaning, and due rather to poor nutrition than to any specific vasomotor effect, but in all of which a more or less rapidly developing and progressing hypotension may be the sign of acute toxic vasomotor insufficiency.

Typhoid Fever.—As a characteristically hypotensive disease, typhoid fever is of special importance in the opportunity it affords of studying and comparing the effects on the circulation of true and reputed stimulant drugs and measures—and there is certainly no pathologic condition, apart from surgical or traumatic shock, in which blood pressure changes are more significant in prognosis than in the typhoid toxæmia. Typically, there is in the course of this disease a slow and regular diminution in the systolic blood pressure parallel with the development of the intoxication, and any break or alteration in this flat curve is of easily recognizable significance. With practically every complication except pneumonia, and with every increase in the severity of the infection, there is a concomitant fall in pressure. With perforation and peritonitis there is an early and striking rise in the period of peritoneal irritation, exactly comparable to that occurring early during laparotomy, and equally certain to be followed by a rapid and profound depression. The indication, in kind and amount, for stimulant drugs, can be derived from blood pressure estimations in the course of typhoid fever with almost mathematical exactness.

In a recent number of the *Journal of the American Medical Association* (May 9, 1903), Crile publishes a preliminary note emphasizing the value of blood pressure measurements in the diagnosis of perforative peritonitis in typhoid fever. In all of five cases observed, he

CHART No. XXII.



Typhoid Fever—Intestinal Perforation.

(1) Level curve of blood pressure for days before the onset of the complication.

(2) Sharp rise, amounting to 36 mm. Hg. in blood pressure, over four hours before the onset of symptoms pointing to the abdomen, and at a time when the abdomen was soft and not tender, and not distended.

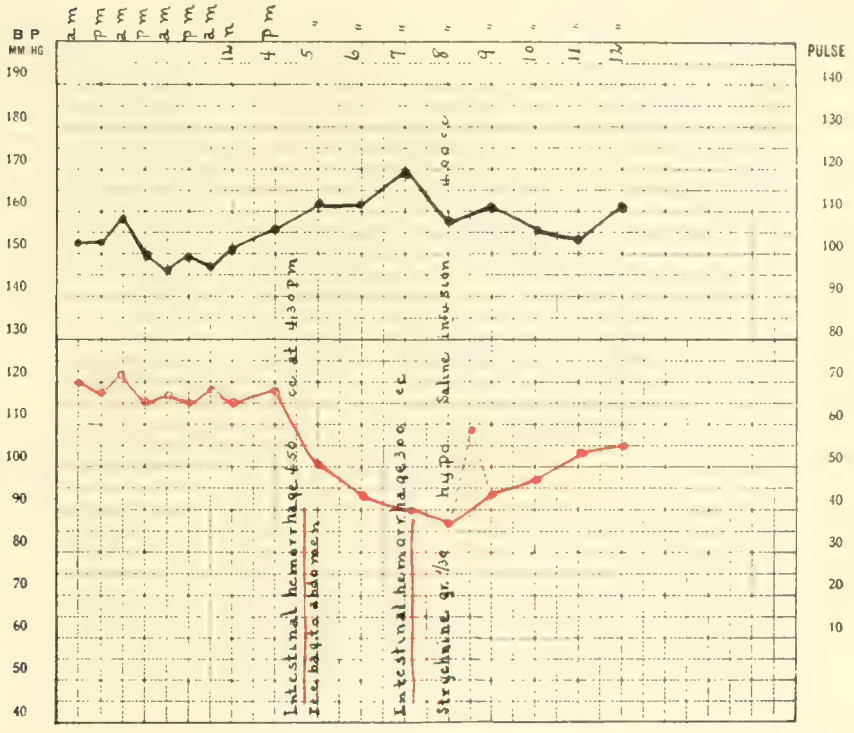
(3) Tendency of the blood pressure to return to a low level as the toxæmia deepened, and splanchnic vaso-constriction was giving way to splanchnic engorgement. Slight post-operative depression.

found that a rise in blood pressure, as estimated by a Riva-Rocci instrument, attended the onset of peritonitis. In twenty cases of peritonitis from other causes, hypertension characterized the pulse, and the abnormally high blood pressures appear to have persisted.

We were led to expect such a reaction on the part of the blood pressure under these conditions almost a year ago, by a suggestion of Dr. Cushing's, who called our attention to the similarity of conditions in ordinary laparotomy (as, for appendectomy) and in the event of perforation of an intestinal ulcer, with consequent peritoneal irritation. In the first instance, the opening of the abdominal wall and mechanical contact of instruments and gauze with the peritoneal surfaces produce typically a sharp rise in blood pressure (Chart III), followed always, if the irritation is long continued enough to produce reflex splanchnic vaso-dilatation, by a fall in blood pressure below its former level. By inference, a similar reaction on the part of the vasomotor center should occur at the onset of any irritation, mechanical, bacterial, or both combined, of the peritoneal tissues—and that the first or hypertensive phase of the laparotomy curve is duplicated by that of typhoid perforation, is evident enough from Dr. Crile's and our own cases. In all of ours, however, this initial rise in blood pressure has been followed by a fall when the peritoneal toxæmia becomes marked and vasomotor paralysis of the splanchnic area has ensued, thus affording a complete parallelism with the blood pressure curve typically obtained in the course of abdominal section. In only one case of typhoid fever with perforation (and subsequent operation) have we been fortunate enough to obtain a long record of the blood pressure for days preceding the onset of the peritonitis, but this case was one of exceptional interest. (Chart XIII.) The patient was suffering from a profound intoxication, was frequently delirious, and had been under stimulation during the whole time of observation; for ten days the blood pressure had ranged between 98 mm. Hg. and 110 mm. Hg., occasionally rising to 115 mm. Hg. immediately after the administration of stimulant (strychnine). There had been slight diarrhœa, no hæmorrhage, and no symptoms of any kind to call attention especially to the abdomen, until in the course of the routine measurements the blood pressure was found one night to have risen within four hours from its previous level to a height of 145 mm. Hg. The patient at that time was in no pain, the abdomen was soft, not distended, and nowhere tender on palpation.

Four hours and a half afterwards the patient, while in a tub, cried out suddenly with severe abdominal pain and was nauseated, and on examination was found to have a tender, rigid abdomen, with very suggestive muscle spasm, and beginning distension. At operation several

CHART No. XXIII.



Typhoid Fever—Intestinal Hemorrhage.

(1) Blood pressure level broken by a sharp fall with the first hemorrhage, with no precedent rise in tension—distinction from intestinal perforation. The fall continues with a second hemorrhage.

(2) Gradual return of blood pressure after the cessation of hemorrage. The physiological return aided, though the curve is not materially altered, by a small dose (gr. 1-50) of strychnine and a small saline infusion (?).

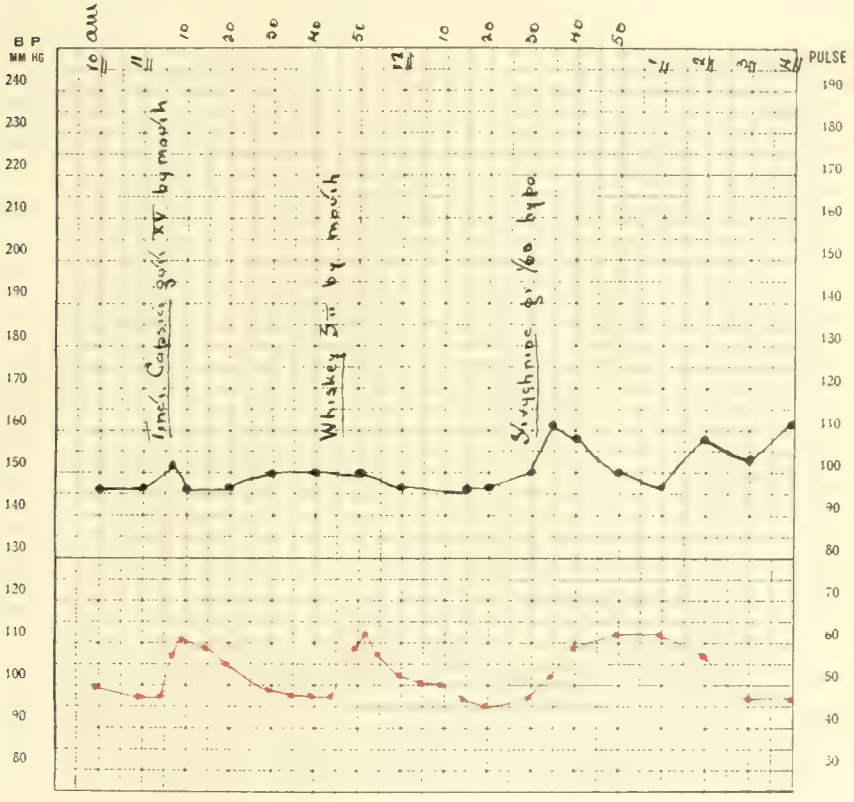
perforations were found low down in the ileum, there was pus in the pelvis, and a rapidly spreading peritonitis; there had at no time been any leucocytosis.

Here, then, is a case in which a rise in blood pressure, produced by the irritation of the peritoneum by foreign matter and bacteria, preceded by a very definite interval the onset of any other sign or symptom which could justify the suspicion of perforative peritonitis. From the nature of its production, as well as on the grounds of the experience of Crile and ourselves, this hypertension must be one of the most constant attending features of intestinal perforation, and in the light of the case cited above, it should, when it occurs in typhoid fever, direct the attention of the attendant as clearly to the abdomen as seat of trouble as even the onset of sudden local pain itself. For no other complication of the disease is capable of producing this decided and sudden rise in blood pressure, with the exception of lobar pneumonia with its very characteristic picture, and the many mechanical and physiological factors that tend to produce hypertension are usually excluded carefully from affecting the typhoid patient, while their action is much less pronounced and of shorter duration than in health. Even the cold Brand bath produces no such rise in pulse tension as the onset of perforation, and its effects are but transient, while the hypertension of peritonitis lasts until toxic paralysis of the intra-abdominal vessels sets in. A sharp and well-maintained rise in blood pressure in typhoid fever, from a previously known low level, is then a valuable sign of intestinal perforation, and is probably of more constant value than the notoriously uncertain increase in the leucocyte count.[1]

[1] How safely the absence of any rise in systolic blood pressure, in a typhoid patient with suggestive abdominal signs, may be relied upon in negativing the diagnosis of intestinal perforation, is very doubtful. Certainly, in a doubtful case, with no peritoneal vertex hypertension of the pulse, it must be of the highest importance to determine the state of irritability of the vaso-motor centre. If the patient's toxaemia is so profound that there is no blood pressure response to central (strychnine) or peripheral sensory irritation (skin), the diagnosis of perforation must be considered quite without reference to the unaltered blood pressure level. We have recently seen two cases of profound typhoid toxaemia, with extremely suggestive abdominal signs, without rising blood pressures. In one case the vaso-motor reflex activity was proven to be unimpaired, and we considered perforation impossible in view of the level blood pressure—operation was insisted upon, and no perforation or peritonitis found. In the other case, one even more deeply toxaemic than the first, no attempt was made to determine whether the vaso-motors were still capable of reacting normally, and at autopsy a perforation was discovered, with early and rather localized peritonitis. We were unable to see this latter case during life, but it seems to us to emphasize the necessity of studying each case thoroughly with regard to the blood pressure, rationally differentiating and eliminating the various hyper- and hypo-tensive factors. Only in this way can blood pressure measurements be made to give reliable indications in any class of cases.

As intestinal hæmorrhage in typhoid fever is often associated with abdominal pain and other features suggestive of peritoneal involvement, and as a large proportion of the cases of perforation of the

CHART No. XXIV.



Typhoid fever (1st day—mild attack)—No previous stimulation.

(1) Tincture of capsicum and whiskey successively producing transient and almost identical pressor effects, and probably acting in the same way, by peripheral (gastric) irritation.

(2) Strychnine producing the long-continued rise in blood pressure characteristic of a true stimulant.

bowel in this disease occurs in connection with loss of blood from the intestine, the value of blood pressure observations in the occasionally necessary differentiation of the two conditions is evident. Intestinal

hæmorrhage is accompanied from the first by a fall in blood pressure, with no precedent rise, and such a change in the pressure curve of a typhoid patient with hæmorrhages excludes the possibility of perforation having occurred. (Chart XXIII.) When, however, in the case of hæmorrhage from the bowel with abdominal pain and other symptoms, there is no fall in blood pressure, but a rise, or a lowering of the pulse tension is preceded by a well-marked and sustained rise, then the probability of the two complications having occurred together is very great. We have never had the opportunity of making blood pressure observations on such a case of combined perforation and hæmorrhage, but the different elements of the case in question are so well known and their effects on blood pressure individually so invariably the same, that it seems safe to predict what the actual curve of the blood pressure would be.

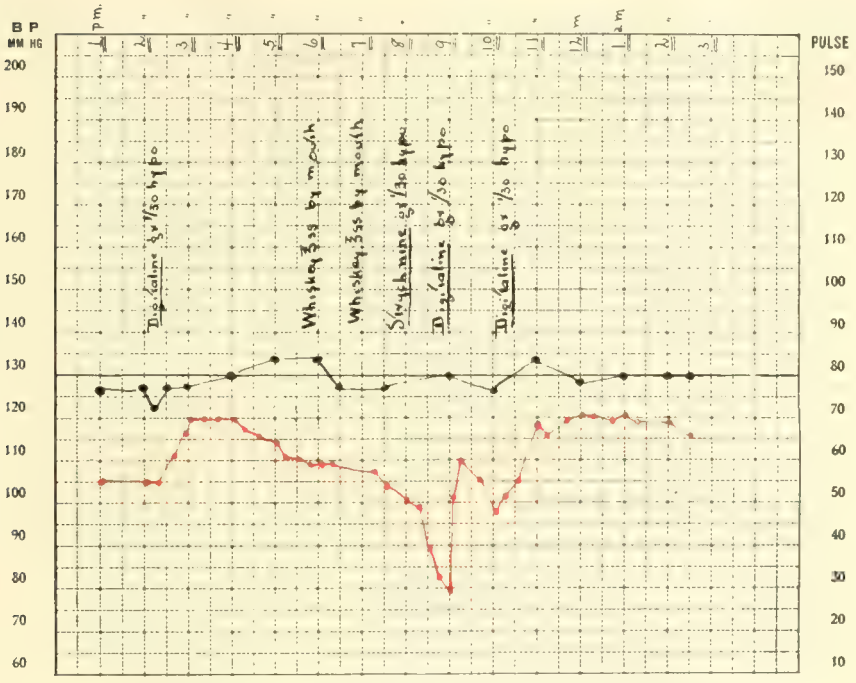
Lobar Pneumonia.—In pneumonia, the disease of all others in which stimulation constitutes the greater part, indeed almost all, of our present treatment, the indication for this treatment in the majority of cases is derived far more from general symptoms than from the character of the pulse. The relative unimportance of the pulse in the prognosis of pneumonia is generally recognized, and it was only to be expected that blood pressure determinations would throw little light on the progress of the disease, and the effect of the usual treatment. With the typical hypertension that exists in this disease, it is interesting that alcohol should have an especially marked effect in lowering the heightened tension, and that strychnia and digitalin, effective as they are in hypotensive conditions in raising blood pressure, should have in general comparatively little discernible effect in this direction in the pneumococcic toxæmia. Except in the event of the dreaded cardio-vascular collapse, blood pressure readings give no very important information in the control of the treatment in pneumonia.

VI. The Comparative Study of General Stimulant Measures.

General stimulation, as a therapeutic procedure, is difficult of exact definition. From the viewpoint of the experimental pharmacologist there are but few drugs which can even in an imperfect way claim a position as universal, or even fairly general stimulants, while the selective action of all other drugs narrows their influence down to a comparatively small number of organs, generally within the same

system. In this sense, it is necessary to define the action of any stimulant drug by a reference to the organ or organs which alone respond to its administration by increased functional activity. The clinician, however, is often forced by the nature of his material to con-

CHART No. XXV.



Typhoid Fever with Toxæmic Features and Hypotension.

(1) Rise in blood pressure following each of several doses of digitalin hypodermically.

(2) Alcohol by mouth and strychnine hypodermically without any beneficial effect on the blood pressure, even during a period of acute depression (threatened collapse).

The patient had received routine doses of whiskey and strychnine for some weeks. The digitalin charted above was the first that had been given.

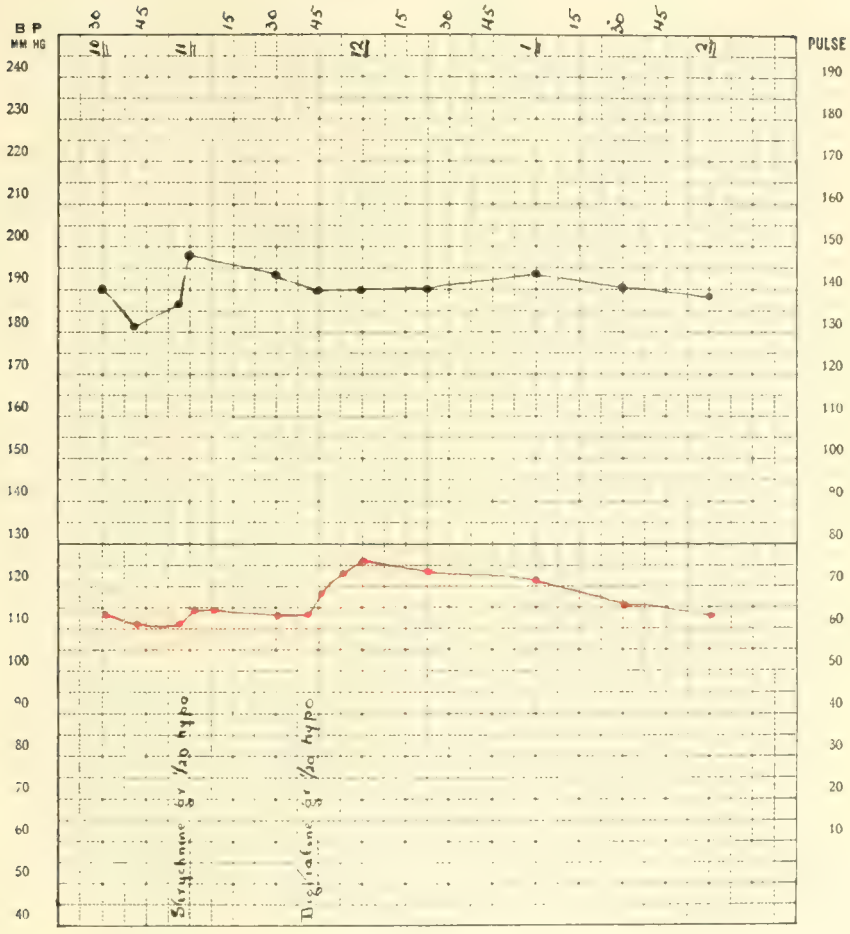
sider the subject in a broader and less accurate way, and while not ignoring the specificity of action on different systems of organs within the body, of various stimulant drugs, would probably agree to the defini-

tion of a general stimulant as a substance whose effect after absorption is to improve the blood circulatory activity, while not depressing, and if possible increasing, the activity of the cerebro-spinal axis. That the effect on the heart and vessels of this class of drugs is the one by which in the main their use is regulated, is evident from the universal practice of prescribing "stimulants" on the indications derived from the pulse. Strychnia and digitalin, alcohol and camphor, are all ordered in the first instance because the pulse feels weak, and perhaps irregular. This cardio-vascular weakening is accepted and recognized as the best guide to reduction of general, as well as exclusively circulatory tone, and as the most reliable signal for the use of supporting drugs.

Having experienced the inexactness of touch judgments in the estimation of pulse force, and realizing the value of the sphygmomanometer in the measurement of that force, it occurred to one of us to observe the effects of stimulant therapy in general on the blood pressure, while serving in the Thomas Wilson Sanitarium during the summer of 1902, and a résumé of these observations has been published recently. It was found in general that the true stimulant drugs all act alike in raising the systolic arterial blood pressure to varying degrees; that the amount and permanency of this change is very fairly proportionate to the betterment in the patient's condition, which may be deduced from the change in general symptoms to be the result of this treatment, and that in all conditions demanding stimulation, and with low blood pressures, the measured changes in pulse tension during and after treatment afford an accurate and a safe index to the efficacy of the drugs employed. These observations have been continued during the past winter in the wards of the Johns Hopkins Hospital, and have fortified the above conclusions. We have studied in some detail the effect on abnormally low blood pressures of all the common drugs generally used as stimulants, in the endeavor to substitute an intelligible measure in their use, in the place of the verbal descriptions of sensations only.

It is not to be thought that we look on every drug that will raise the blood pressure as necessarily a stimulant, or on measured hypotension as the only indication for stimulation. But we believe that the majority of men in practice judge of the effect of their stimulants in great part by their tendency to improve the pulse force, and that pulse force in this sense can always be taken to mean the touch

CHART No. XXVI.



Fatal Relapse in Typhoid Fever—Profound Toxæmia.

(1) The patient had received strychnine regularly for three weeks—the dose charted is without effect on the blood pressure.

(2) Digitalin had been given but three times before the above record was taken. The dose charted produced a definite, though not very great, increase in blood pressure.

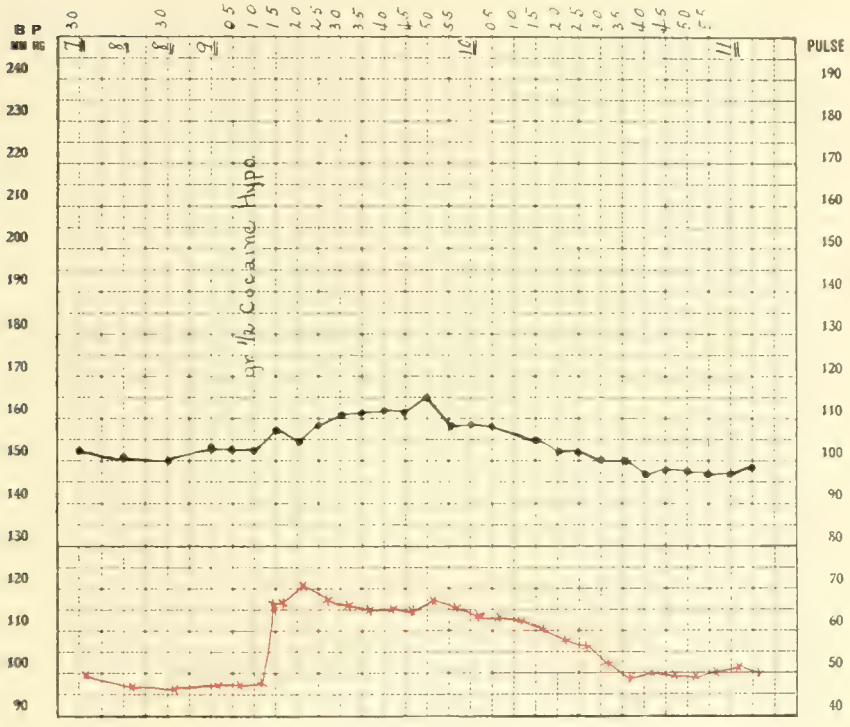
judgment of systolic intra-arterial tension, while the finger, though the most ready, is in many conditions of the circulation the most deceptively and grossly inaccurate of all sphygmomanometers. That all stimulant drugs under favorable circumstances do raise abnormally low blood pressures, will be soon evident to anyone who will investigate the matter clinically, or will look into the physiological literature on the subject—while a drug that is generally powerless clinically to raise the systolic tension will not be found classed among stimulants by the laboratory pharmacologist.

In so far, then, we believe systolic blood pressures to be a guide to the employment of stimulant drugs, and the most accurate clinical test of their efficiency. Our results have brought out, as might be anticipated, no revolutionary conclusions, and in the few points where our methods lead us to differ in detail with the principles of practice generally received, we have attempted to apply both physiological and clinical criteria to their analysis. Our observations have been made on cases receiving routine or occasional stimulation in the wards, the large part of these patients suffering from severe typhoid infections, but controlled by records of many different sorts of cases, marantic, toxic, and infectious, in which stimulation was practiced. They are the records of the effects of stimulants in hypotensive states exclusively, and we have not discussed the relatively rare conditions, of which acute pneumonia is the only common example, in which free stimulation is not necessarily given for the relief of hypotension.

Strychnine.—Strychnine is of all drugs the one whose stimulant properties are most generally recognized and employed in practice, by the physiologist and clinician alike, and the study of its clinical influence on blood pressure in the systolic phase affords a type for the comparison of all other drugs of this class. Given hypodermically in doses of from 1-60 to 1-10 of a grain, it produces a rise in blood pressure the onset of which is somewhat delayed as compared with that sometimes seen following the administration of alcohol by mouth, whose increase is not so abrupt, but which is maintained for a much greater length of time, varying in average cases from one to four hours. This rise in blood pressure is distinctly seen to be accompanied, in the majority of patients carefully observed, by an improvement in the patient's general condition, and we have never observed a patient growing obviously more toxic, or with mental symptoms, if present, showing any aggravation, during a period of increase in the force of the pulse fol-

lowing this drug When the routine administration of strychnine has continued for from 8 to 12 doses, the individual doses often fail to produce any marked immediate rise in the systolic blood pressure, but in such cases if one or two doses are omitted and the pressure is care-

CHART No. XXVII.



Post-Operative Depression.

Rise in blood pressure produced by cocaine (central stimulation).

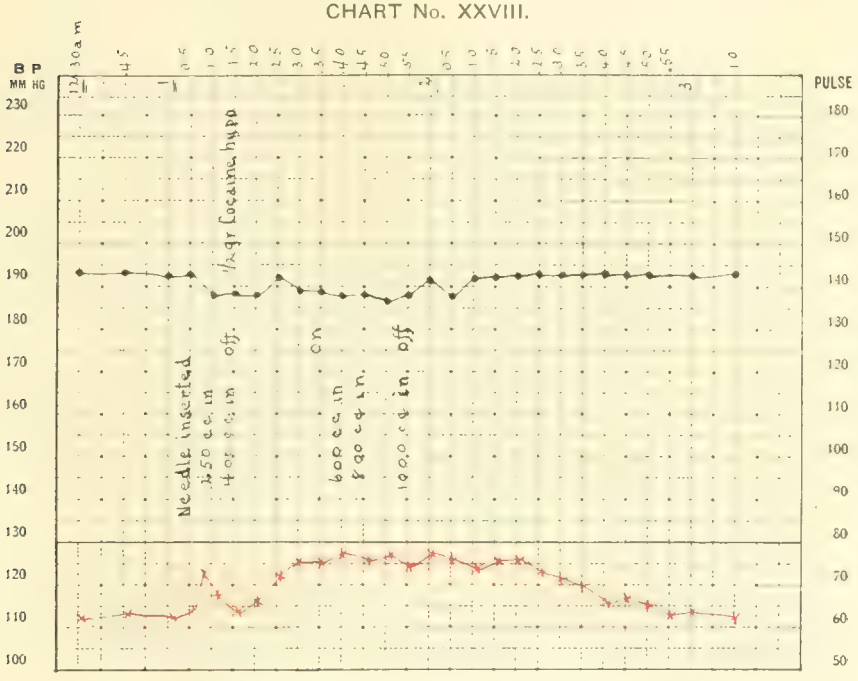
fully followed in the interval, it will be seen that there is a progressive fall in the blood pressure in the absence of stimulation, the previous level being again restored when the drug is renewed. On the whole, strychnine is by far the most satisfactory cardio-vascular stimulant for long-continued routine administration, the maintenance of a satisfactory blood pressure level, free from intervals of depression, being most easily accomplished by its use in appropriate doses. (Charts VII, X, XXIV, XXVI, XXXIV.)

When in the absence of any previous stimulation, or where general stimulant treatment has been inadequate, it is desired to quickly raise a markedly lowered blood pressure, strychnine often has a most dramatic effect. In such circumstances large doses are required, and 1-10 of a grain, or 1-20 soon repeated, is not an excessive amount for hypodermic administration in adults. (Chart VII.) Under such large initial doses the blood pressure may be seen in most cases to rise rapidly from a disturbingly low level to one approaching the normal—a rise from 80 mm. Hg. to 120 mm. Hg. being by no means extraordinary after 1-20 of a grain. The gain is not merely transient, but will endure for an hour or more if the patient be not moribund; at the end of that time, or when the pressure begins again to fall, a much smaller dose will usually suffice to check any further decline, and then the regular administration of small doses, in connection with digitalin if necessary, will maintain a safe level. We have been much impressed by the benefit obtained from these large initial doses, provided the patient can be carefully watched and that constant comparisons can be made on the pulse. Far better results can be obtained in this way than by ordering a routine dose to be given on the rather indefinite indication of a "weak pulse" and then leaving the patient to pass, say, the eight hours of the night without further control.

There are three great groups of cases in which the hypodermic administration of moderate and large doses of strychnine fails to raise the arterial blood pressure, and the same remarks will apply in general to the use of digitalin hypodermically. At one extreme are the moribund, hopeless cases, in whom the blood circulatory apparatus is near to its final breakdown, and is incapable of any further forcing, the failure to react characteristically being one signal of the approaching end. On the other hand we have cases not really in need of stimulation—relatively normal individuals whose heart and vessels are in a state of healthy tone, and in whom drugs have far less effect on the blood pressure than purely physiological demands for increased pulse energy. As a mean between these two groups stand the cases already mentioned in whom continued stimulation at regular intervals maintains the blood pressure at what we may call the stimulation level of the case—a numerical or graphic representation of the best possible output of energy in the circumstances. In all these cases we expect to find little or no immediate effect on arterial blood pressure produced by any single dose of stimulant drugs.

Digitalin.—Digitalin hypodermically in doses equal to those of strychnine, is more certain than the latter in its pressor effect. (Charts XXV, XXVI, XXXIV.) The action of digitalin in raising the systolic pressure is manifest rather earlier than that of strychnine, and is in general more rapid in reaching the maximum, while the effect of a

CHART No. XXVIII.



Slight Depression following Abortion.

(1) Rises in blood pressure, during continuance of saline infusion, parallel with degree of local tension and irritation—manifestly a peripheral reflex effect.

(2) Similar transient effect of manual examination of the abdomen—splanchnic compression (?), local irritation, and pain.

single dose is usually not so long-continued. Frequently a case which fails to react to any great extent to strychnine will show marked and rapid improvement when digitalin is given, and in such cases particularly the combined exhibition of the two drugs will secure the immediacy of the desired result, and ensure its permanency. With digitalin,

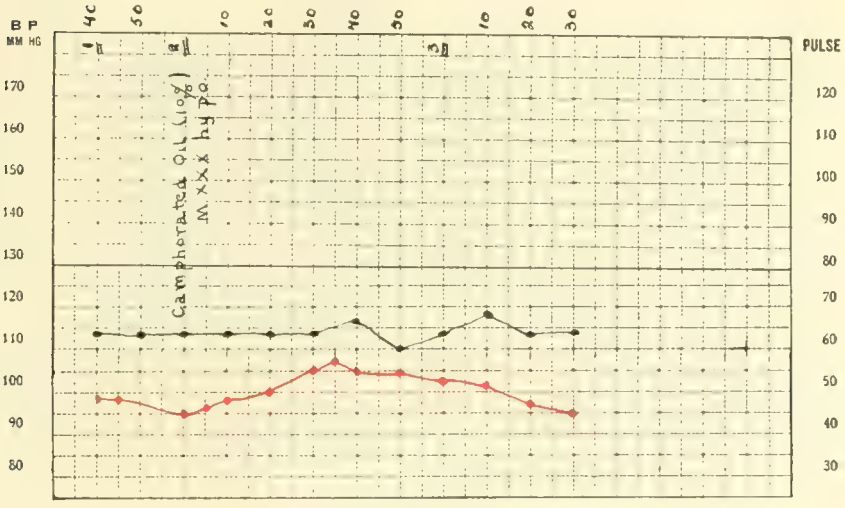
as with strychnine, the initial administration of 1-10 grain doses has proven often of great value. We believe that with a patient in a condition of profound depression, as manifested among other things by a low blood pressure, if we are justified in using stimulants at all, we are bound to do so until we produce some beneficial effect, if it is at all obtainable. Just as the only limit to the dosage of morphia given for severe pain should be the amount necessary, however large it may be, to give relief, so in parallel case, the only limit to the amount of strychnine and digitalin to be given in a case that calls for them at all, should be that quantity which will have the best effects. There seems little doubt that these effects, on the organism as a whole, are best estimated in the conditions here referred to by the state of the circulation, and the blood pressure, though not the only one, is one of the best indications of this state, and certainly the one above all others whose directions can be most accurately followed.

It is not suggested that a desperately ill patient who fails to react to a tenth of a grain of strychnine or digitalin should be subjected to the injection of ¼-grain doses, though these would probably have as little effect as the unavailing tenth. But we are convinced that if by means of continuous blood pressure measurements we keep in close touch with the state of the circulatory apparatus as a whole, we may safely and advantageously use free initial stimulation, with the certainty that we will be acting more intelligently by the patient, and with the probability that he will in the long run receive less drugging than if it had all depended on immeasurable touch perceptions. It is not claimed that the initiation of stimulant treatment should depend solely on the height of the patient's blood pressure—some patients with normal pressures needing stimulation, and others with relatively low pressures (100-115 mm. Hg.) being well able to get along without it. The indication for this form of treatment must rest, as always, on the physician's judgment and the correlation of a number of facts, and clinical common sense can never be replaced by a machine with a numbered scale. But the Riva-Rocci instrument adds a valuable class of facts to those obtainable in other ways, and makes their evaluation more precise, while when stimulation has once been undertaken, it adds an otherwise unknown definiteness to our therapeutic control.

Cocaine.—Cocaine hypodermically has a distinct pressor effect on the circulation and seems to act in the manner of a true stimulant.

(Chart XXVII.) We have used it especially in hæmorrhagic and post-operative cases of hypotension, and in these, at any rate, it seems worthy of a wider use than it at present receives, in connection with strychnine and digitalin. Doses of from one-quarter to one-half a grain produce an almost immediate rise in blood pressure of from 10 to 20 mm. Hg., which is maintained for from one to three hours, and which, though we cannot here speak of toxic cases, is as certain in

CHART No. XXIX.



Urœmia with Coma.

Hypotension in collapse a few hours before death.

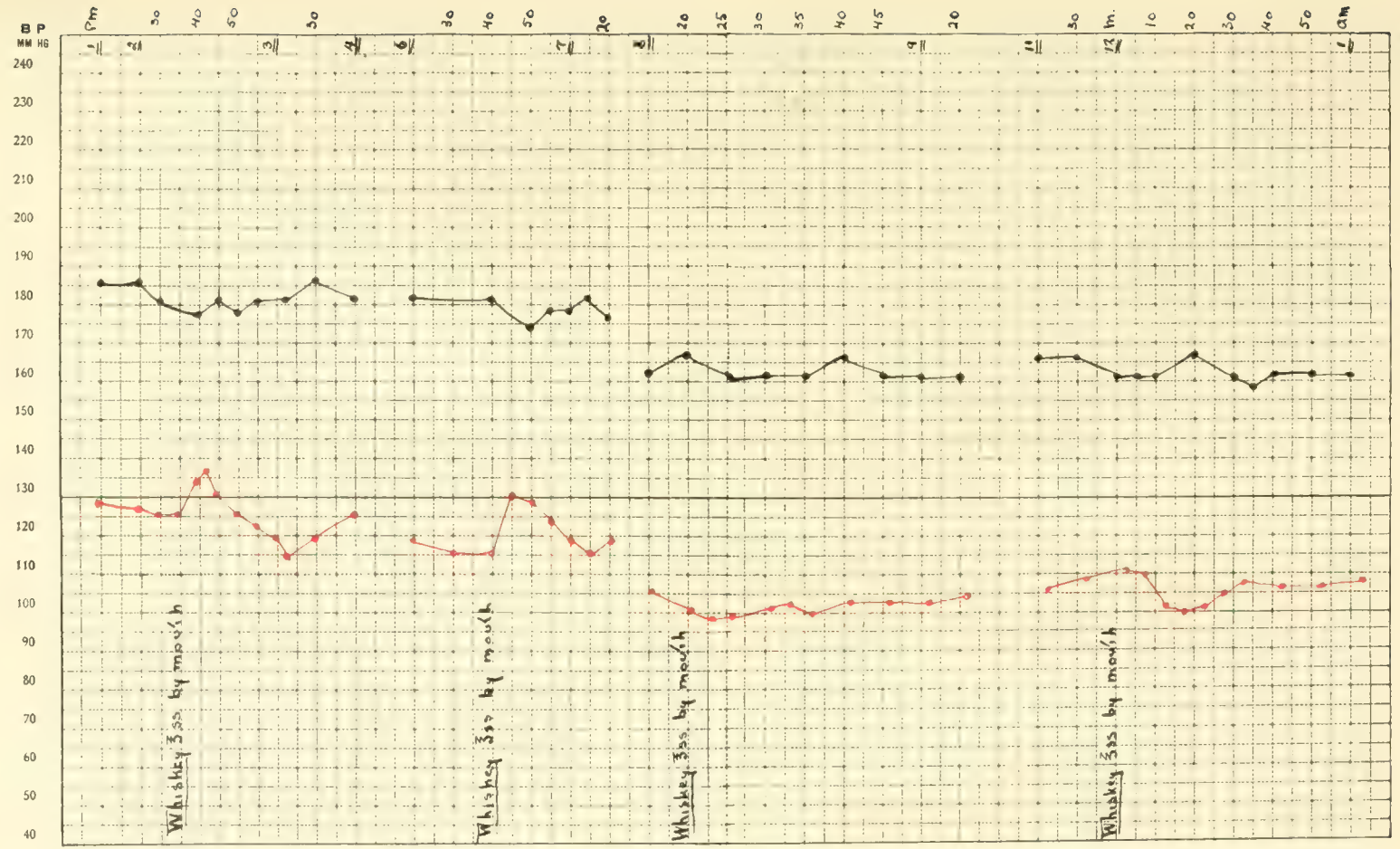
Camphor hypodermically followed by a slight and gradual (slow absorption) rise in blood pressure, without change in the pulse rate. Strychnine in large doses failed to raise this patient's blood pressure—digitalin was not given.

its occurrence as that following strychnine. Especially valuable is the administration of cocaine in connection with saline infusions, thus not only avoiding the occasionally harmful reflex effects of local irritation from the latter, but giving them a positive stimulant value which they do not otherwise possess. (Chart XXVIII.)

Camphor.—This drug is especially valued by French writers, a stimulant action being ascribed to it which is said to be mainly cardiac. We have studied its effect on blood pressure in a few cases of

hypotension, and have found it generally inert where strychnine, digitalin or cocaine exerted marked pressor effects. Its regular use in the long-continued depression of typhoid fever, for instance, does not show any appreciably beneficial effects on the pulse, such as is obtained by the use of the other drugs cited; in other words, it does not increase general vasomotor tone. When, however, low blood pressures are associated with and dependent upon a weakened cardiac systole, as in cardiac loss of compensation with hypotension, or in acute toxic dilatation, camphorated oil hypodermically in doses of from ten to twenty minims of the ten per cent solution produces a definite rise in systolic blood pressure. (Chart XXIV.) This is never so marked or so permanent as that following digitalin or strychnine, and gives no ground for its preference to either of those drugs. It ranks, of course, far below digitalis as a purely cardiac stimulant, and possesses the relative disadvantage, when compared with that drug, of not influencing the vasomotor center or periphery. The indication for its use seems amply covered by digitalin, and the combination of the latter with strychnine makes the administration of camphor as a stimulant quite unnecessary. Where a toleration has been established to digitalis, camphor might perhaps be substituted for it beneficially during a period of intermission of the more powerful drug.

Alcohol.—Alcohol occupies a unique position in any system of therapeutics. The popular use of the term stimulant, which when unmodified seems always to refer to ethyl alcohol in some form, may have unconsciously modified the more strictly professional conception of its properties, and have created a prejudice in favor of this use of the term. However that may be, physiological teaching to-day ascribes no virtue to alcohol in its action on the tissues independent of its possession of combustible atom groups, and the most painstaking research in the laboratory fails to show that it has any cardio-vascular stimulant properties whatsoever in the scientific sense. With its congeners, ether and chloroform, it is from the first, after the moment of its application to an absorptive surface, directly depressive in its action on all the nervous tissues, and the science of pharmacology has brought forward no stronger chain of evidence than that depriving alcohol after ingestion of all true stimulating qualities as regards the circulation. In the face of this evidence, the fact that the mass of clinicians still cling to a belief that places this drug in the same general category with strychnine is surprising enough, and gives special



Pneumonia (period of defervescence—slow crisis) with Moderate Developing Hypotension.—Blood pressure effects of four moderate doses of alcohol within the same twenty four hours. (1) The first two doses of alcohol produce a temporary pressor effect—the result of irritation of the gastric mucosa. (2) The third dose is without any effect on the blood pressure. (3) The first dose and the last are followed by a slight fall in blood pressure—illustrating the depressant action of alcohol after absorption. (4) The pulse rate is unaffected by any one of the doses

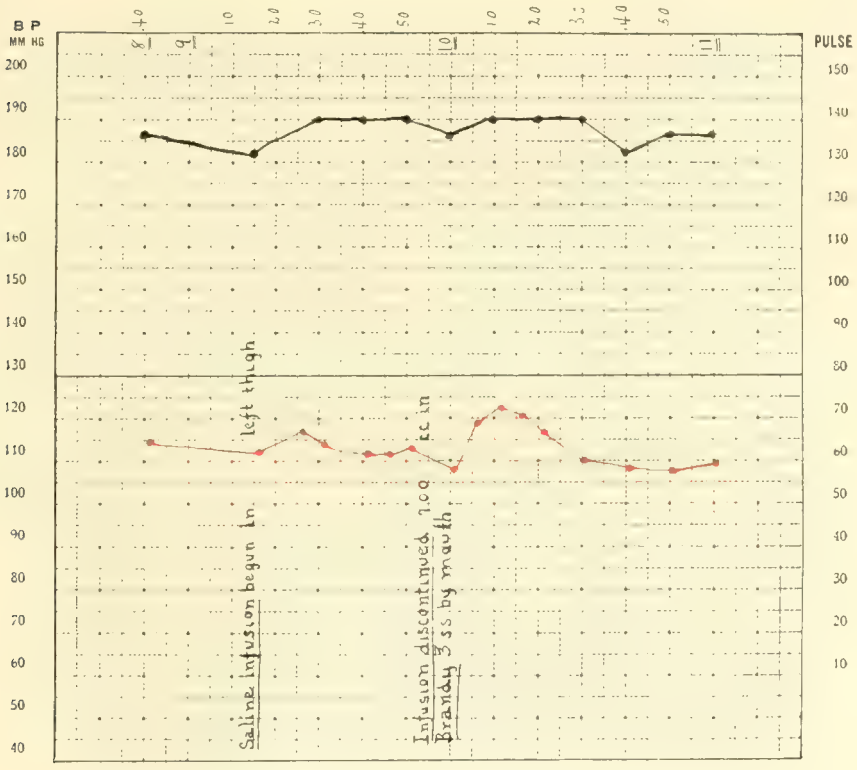
interest to any method that will enable us to apply physiological principles to clinical material in the study of this discrepancy of opinion.

The indications for the use of alcohol, as followed in the wards of the Johns Hopkins Hospital, may be summed up by the term toxæmia. It is rather the accumulation of the many signs and symptoms of that condition, than any one particular symptom, that guides the ordering of the drug, and the character of the pulse cannot be said to be the most prominently emphasized of those signs. It is, however, so generally found in the literature that the administration of alcohol is controlled by the state of the pulse, and the opinion is so persistently expressed of its value in increasing cardio-vascular activity, that an accurate study of its actual effects on the systolic energy of the blood circulation cannot but be of value. The results of such a study are strictly in accord with the conclusions drawn from the latest pharmacological evidence, and seem to force the opinion, which is by no means a new one, that the unquestioned good effects of alcohol in certain infectious and toxic conditions must result solely from some obscure and as yet unexplained alterative influence on metabolism—as by favoring the destruction of toxins, or rendering them more suitable for elimination. This side of a very interesting question we have not attempted to touch upon.

After absorption into the body, alcohol never increases the force of the pulse. Any rise in systolic blood pressure observed after its use results wholly from a reflex action, through irritation of the afferent nerves in the mucous membrane over which it passes, or in the skin if it be given hypodermically. (Chart XXXI.) Alcohol may have no effect whatever on the blood pressure, even if given in large doses, but characteristically, and in the majority of patients who have not established a tolerance to its action, it lowers the pressure distinctly, and this fall is much more permanent than any preliminary irritative rise that may immediately follow its ingestion. (Chart XXX.) Given hypodermically, the effect on the blood pressure is the same as when taken by mouth or rectum—nil, if local irritation be avoided, always depressive after absorption. The pulse after alcohol shows often, though not uniformly, a change in character as appreciated by the touch—a change conveying the impression of increased force, and which is apparently the basis of all claims for the true stimulant action of the drug. This change in palpation character always occurs during the period of depression of the intravascular tension, is due appar-

ently to an increased difference between systolic and diastolic pressures, and so may evidence a fall in diastolic pressure even greater than that which occurs in systolic pressure. As there is no change in the pulse rate produced by alcohol after absorption, the total work in

CHART No. XXXI.



Typhoid Fever, Relapse, Marked Toxæmia with Coma Vigil.

(1) Infusion of salt solution at body temperature and under little tension producing no effect on the blood pressure—absence of both peripheral irritative reflex and true central stimulation.

(2) Alcohol by mouth producing an immediate but transient rise in blood pressure, without change in pulse rate.

blood propulsion is less under its influence than before, and the very pulse change that expresses this diminution of cardio-vascular activity is habitually misinterpreted in judgments based on touch alone. It

may be, as Gutnikow claims (19), that the circulation of the blood is carried on more advantageously in some way after the ingestion of alcohol, just as nitroglycerin may steady a failing heart by relieving the excessive resistance against which it works. But this action is surely not a stimulant one, and alcohol should no more be classed among the stimulant drugs than should the depressant nitrites.

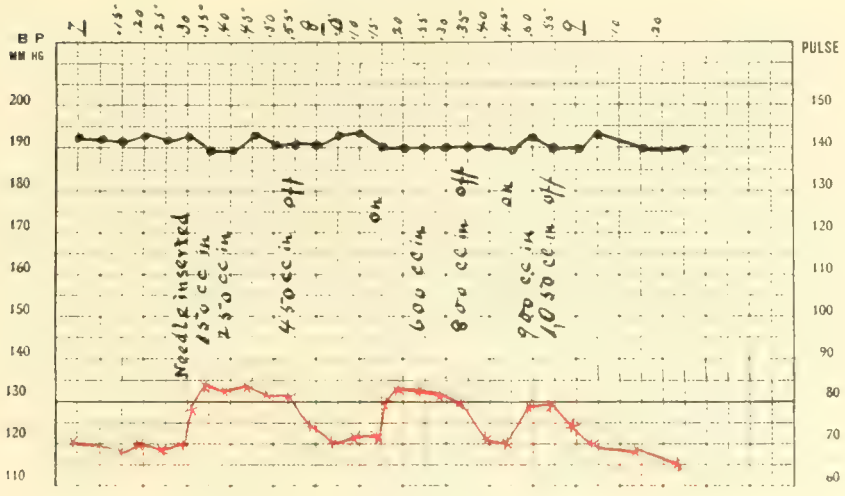
That the preliminary and inconstant rise in blood pressure not infrequently seen following this drug is solely an expression of local nerve-end irritation, necessarily follows from the following facts. When given by mouth, alcohol produces no greater or more permanent rise in blood pressure than does the administration of much smaller doses of such irritants as tincture of capsicum. (Chart XXIV.) A similar and equally transient rise follows the hypodermic injection of undiluted brandy in the usual doses. On the other hand, as large amounts as 30 cc. of 90 per cent alcohol may be introduced beneath the skin without producing any rise in the blood pressure, provided the alcohol be diluted sufficiently with normal saline solution to eliminate the irritant effect on the tissues that it has in concentrated solutions. True stimulants, when administered in this manner, elicit, on the contrary, a positive vasomotor response.

Recently Binz (20), in a consideration of the therapeutic use of alcohol in the light of the work of the last ten years, has made another argument in defense of its stimulant properties. The greater part of his two papers is taken up with the report of experiments on the effect of alcohol on the respiration in animals and man, and with a discussion of the drug in its relation to metabolism. The particular interest of Binz's communication, however, lies in his report of experiments in man showing a definite rise in blood pressure following the ingestion of alcohol in relatively dilute form (sherry wine). He is properly cautious in attributing this rise entirely to increased cardiac energy, but says that "far from having shown paralysis" (of the cardiovascular mechanism as a whole) "our experiments demonstrate that the total effect of alcohol . . . is in the opposite direction," i. e., that alcohol is a true stimulant in moderate doses. He gives no data as to the number or the nature of the cases observed, and seems never to have used the more concentrated spirits, though this point is not specifically mentioned. Very meagre details only are given as to the conditions under which his observations were made, and the actual figures of but three experiments are mentioned. In all of these a rise

in blood pressure of from 15 to 50 mm. Hg. is reported as following the ingestion of less than two ounces of sherry, and in one case a rise of 30 mm. Hg. persisted for "three or four hours!"

These data, inconclusive as they are, seem certainly to be in direct contradiction not only with our own observations, derived from the study of the effects of alcohol in nearly a hundred cases, but with the views of Abel (21) and the other experimental pharmacologists. Abel, after a review of the literature, and reasoning from his own exhaustive

CHART No. XXXII.



Saline Infusion.

Post-partum hemorrhage after physiological return of blood pressure to a practically normal level. Rises in blood pressure dependent on intermittent increase in local tissue tension at the point of injection. Pulse rate unaffected.

experiments, says:

"Alcohol as such, introduced into the circulation with the avoidance of local irritation, is not a circulatory stimulant. In moderate (therapeutic) quantities, it has no effect on the heart itself, on the walls of the blood-vessels, nor on the arterial blood pressure. When a change in the blood pressure does become apparent, it is always in the direction of a fall, and not of a rise. In respect to its inherent action when once in the blood and tissues, alcohol must be classed with the

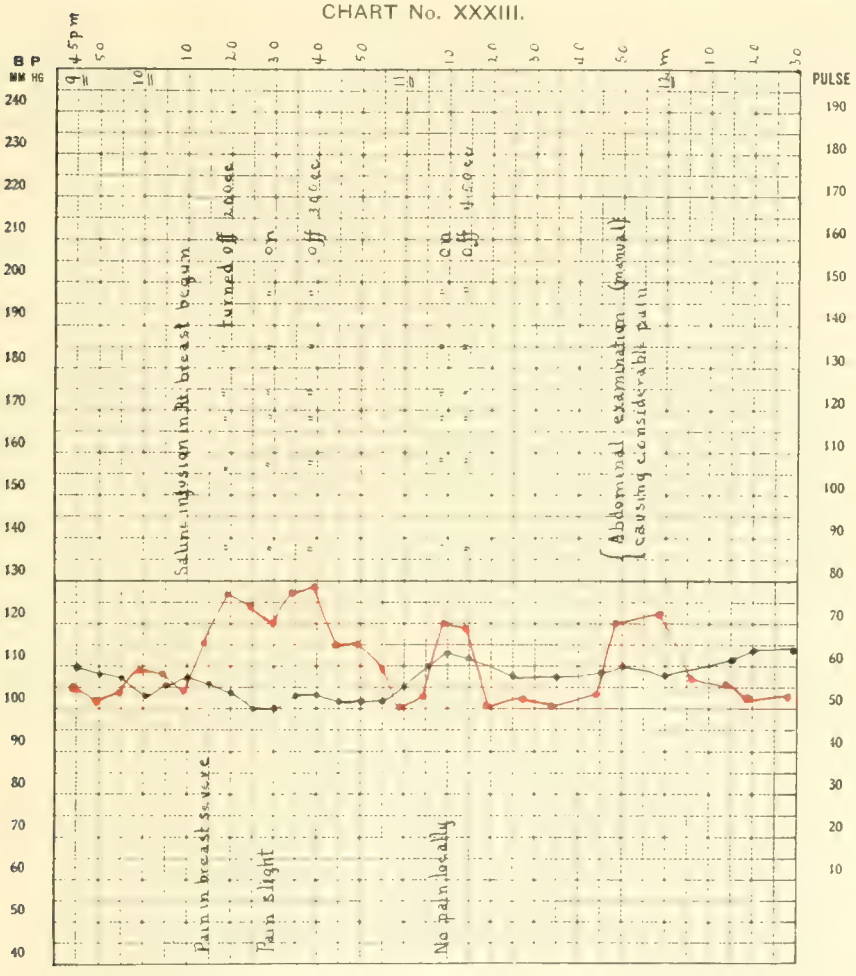
anæsthetics and narcotics—its indirect influences (physical and irritative) must not be allowed to hide its true character, which is always that of a depressant."

It does not appear that Binz, in his report at least, has established enough controls to determine the part played by the indirect, local and mental, effects of the ingestion of wine in his experiments. It is also remarkable that he should have observed after moderate doses of sherry, rises in blood pressure which in duration and degree equal, and in one case at least exceed, the best results we have seen following the hypodermic administration of maximal therapeutic doses of strychnine in the most favorable toxic cases. The reason, however, for the discrepancy of our results is not far to seek, even if it be certain that Binz's and our own observations were obtained under otherwise exactly comparable conditions. His determinations of blood pressure were made wholly with the instrument of v. Basch, whose readings are notoriously inaccurate, and are not in fairness at all comparable, or worthy of equal authority, with the readings of a sphygmomanometer of the Riva-Rocci type. Personal experiment with the v. Basch instrument is enough to show that its readings may vary within limits that fully include the extremes noted by Binz, and quite independently of any change in actual blood pressure. It is regrettable that the comparatively small number of studies of blood pressure in the clinic that have been reported, should have their value so depreciated by the use of unreliable instruments, and the consequent absence of comparable data.[1]

Saline Infusions.—(I) The subcutaneous administration of large (500-1000 cc.) quantities of normal salt solution is undoubtedly very widely used in a great variety of conditions where more or less heroic "stimulation" is considered necessary. Given in this way, it is supposed to have a twofold action, in the first place acting mechanically by increasing the total volume of the blood, "filling up the vessels,"

[1] Since the preparation of this paper, Cabot has published the results of his important research on the action of alcohol in febrile conditions (B. M. and S. J., July 23, '03). His conclusions anticipate and amplify those to be derived from our own less extended observations. Cabot finds, in sum, that "neither the maximum nor the minimum blood pressure showed any variations that could reasonably be referred to the action of alcohol in therapeutic doses. So far as was determined by the methods and the cases used in this research, the action of alcohol upon the circulation was nil." Our findings, and their interpretation, differ from Dr. Cabot's only in details; his and our own general conclusions are, in regard to the fundamental principle involved, in perfect harmony with the views of the laboratory pharmacologists.

and secondly by producing a directly stimulant effect on the heart and vessels and their nervous controlling mechanism: while the whole effect



Post-partum Hæmorrhage with Hypotension.

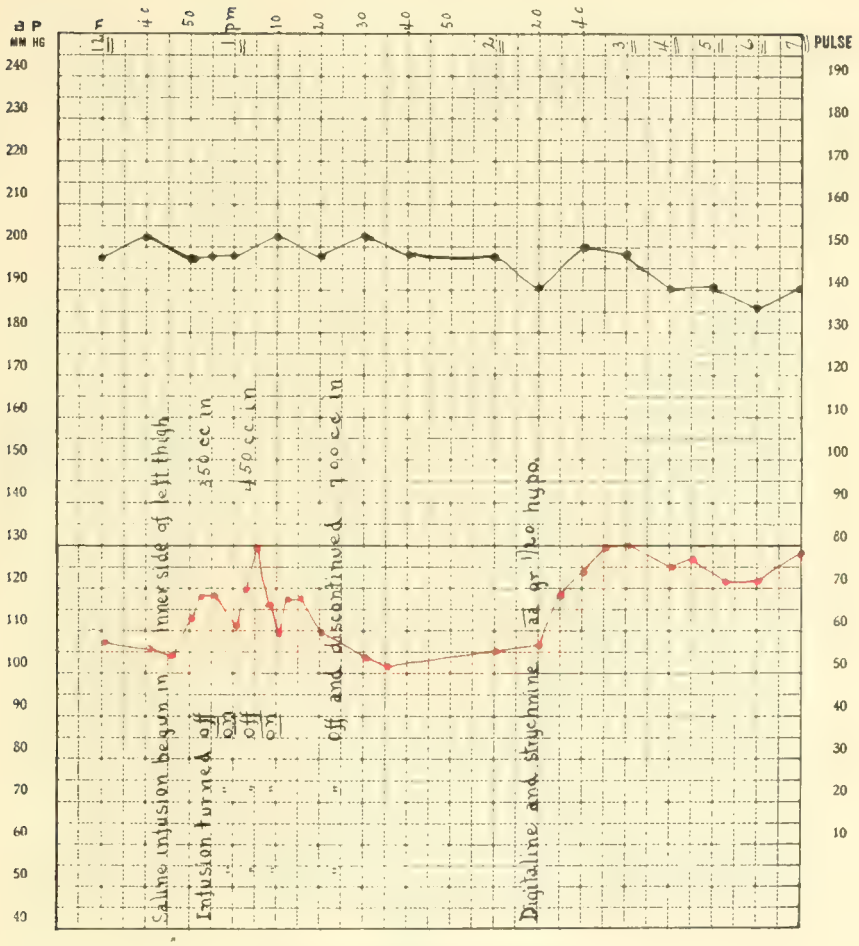
Transient pressor effects produced by intermittent tissue tension (saline infusion) and by painful abdominal examination.

of the infusion is supposed to be very much more beneficial than that of the ingestion of an equal volume of fluid and salt by mouth or rectum. If this is so, if saline infusions possess this plethorizing and

stimulant power, then we should expect their use to be followed constantly by a definite rise in blood pressure, whose degree would be roughly proportional to the amount of fluid absorbed. Accordingly, during the past year we have obtained continuous records of the systolic blood pressures in a large number and variety of cases before, during and after the administration of salt solution, with the object of confirming if possible by physiological methods the current clinical opinion.

We have found saline infusions to be without cardio-vascular stimulant effect. In but one of over a hundred cases other than hæmorrhagic has the infusion produced a rise in blood pressure that might fairly be attributed to the action of the salt solution after absorption, and that was in a much dessicated old person who had long suffered from chronic intestinal obstruction and who had taken and retained but minimal quantities of fluid for a period of months. In none of the others did an appreciable rise in pressure accompany or follow the infusion of even very large quantities of fluid, except as it was due to purely local and peripheral nervous causes. What occurs when the normal salt solution is introduced into the tissues, under moderate tension and at a temperature somewhat above that of the body, is about as follows (see charts), the reaction described here applying only to patients whose hypotension is not due to shock. With the introduction of the needle, especially if it causes much pain, there is often a temporary rise, rarely amounting to 10 mm. Hg., and quickly obscured by other features of the curve. As the solution flows into the tissues, and *pari passu* with the development of local tension, the blood pressure may be observed to rise, often to a very considerable (20 to 40 mm. Hg.) height above previous readings, and the pulse becomes palpably stronger and fuller, producing the illusion of a "filling" of the vessels. This rise in tension is rapid at first, then slower, and usually persists for a little at its maximum, and then begins slowly to fall, the blood pressure returning to or about its previous level in from fifteen minutes to half an hour. This if the infusion be continuous. If it be intermittent, and the flow of the solution be stopped from time to time and resumed after a short interval, the blood pressure will be found to rise and fall as the fluid is introduced or interrupted. In a continuous infusion of, say, one litre of fluid occupying an hour's time, the blood pressure will return to its usual level long before the infusion is completed, its fall following the early rise occurring even during a time when the local tension in the tissues is great and in-

CHART No. XXXIV.



Profound Typhoid Toxæmia.

(1) Transient pressor effect of a warm (105°-110°) saline infusion given rather rapidly. Blood pressure curve similar to that following any peripheral irritation, varying with changes in local tension as solution is turned on and off, and returning to the previous level before the termination of the infusion (anæsthesia by infiltration?).

(2) Rise in blood pressure lasting four hours produced by a combined dose of digitalin and strychnine—true central stimulation.

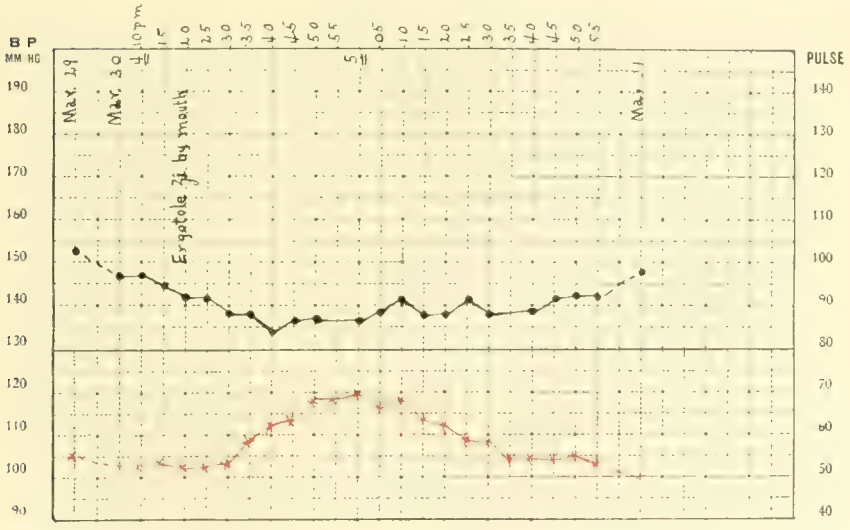
creasing. If cocaine be added to the infusion fluid, the early rise in blood pressure is absent, a slower and more permanent (properly, stimulative) rise being produced after absorption by the cocaine. No rise in blood pressure is observed during the hours of the absorption of the salt solution into the general circulation, that is, there is no late stimulant power possessed by sodium chloride and water, and it remains only to explain the early changes in blood pressure occurring at a time when absorption can only have begun. (Charts X, XXVIII, XXXI-XXXIV).

The facts seem to permit but one interpretation—that the effects noted above are wholly due to a peripheral reflex action on the vasomotor center. These blood pressure effects can be duplicated by any form of intermittent or continued peripheral nerve irritation, and are exactly similar to the changes in pressure accompanying pain, whether spontaneous in the patient or produced by manipulation or minor operation. If any further proof were needed to demonstrate their reflex origin, apart from the exact parallelism that exists between the degree of local tension produced by the infusion and the amount of the concomitant rise in blood pressure, it is afforded by a consideration of the fall in blood pressure that occurs before the completion of the infusion and when the local tissue tension is yet increasing. This fall is very evidently due to an infiltration anæsthetic effect, to a paralysis of the local afferent nerve endings by compression and imbibition of fluid, and so to an interruption at the periphery of the reflex arc whose activity conditions the whole transient pressor effect of saline infusions. This interruption can be equally well accomplished by the addition of small quantities of cocaine to the salt solution, when the characteristic transient "spike" elevations in the blood-pressure curve will be eliminated, and a prolonged elevation obtained at a later period of the curve, when the true central stimulant has had time to enter the circulation and affect the medullary centers. Moreover, if the temperature of the fluid injected be at or very near that of the body, and if it be allowed to run very slowly, so as not to produce a large or tense tumor, there will be absolutely no rise in blood pressure accompanying it.

(II) Turning to another aspect of the question: shock is a condition characterized by loss of vasomotor tone, and in which certain very constant changes occur in the systemic blood pressure. Regarding shock as a state produced by the effect of excessive peripheral stimuli on the medulla, whether that effect is one of exhaustion or of inhibi-

tion, it is obvious that further peripheral irritation must always tend to accentuate the shock, and to still further exaggerate its essential feature —low pulse tension. That this is a fact, is a matter of general clinical acceptance, and the first point in the treatment of shock is everywhere recognized to be protection from outside disturbance, and the ensuring of rest and quiet. Now if frequent blood pressure observations be taken on a shocked individual while undergoing manipulation, or during operation, it will be found that every procedure capable of acting

CHART No. XXXV.



Rise in Blood Pressure Produced by Ergot (Peripheral Vasomotor Stimulation).

as a sensory irritant, every section of a nerve not previously cocainized, will be followed by a fall in blood pressure, that is, by a deepening of the shock (22). This fall may be preceded or not by a very temporary rise in blood pressure, but the final and typical reaction of blood pressure in shock to peripheral stimulation is depressive. Normal individuals, under the same stimuli, feel pain subjectively and show a reflex rise in blood pressure; those in shock are usually conscious of but little pain and their blood pressure shows the inverse, or negative, type of response to that obtained in health. In such patients stimuli that would barely affect the vasomotor balance of a normal organism often produce depressive effects apparently quite out of proportion to their dynamic value.

In shock, then, if our opinion as to the action of hypodermic saline infusions on the circulatory system be just, this measure should prove of little value, and considering the nature of shock and the fundamental influence of low blood pressures in the production of its clinical picture, whatever merit infusions may possess in this condition should be very accurately determinable by means of continuous measurements of pulse tension while the infusion is going on. Quite in accord with observations in many other conditions, our cases show that normal salt solution introduced beneath the skin has absolutely no effect on the blood pressure of individuals in shock, provided, first, that the solution be at body temperature, and secondly, that its injection be so slow as not to produce appreciable tension in the subcutaneous tissues. If the solution be administered in the usual manner, however—warm, and rather rapidly than slowly—the blood pressure shows changes at an early stage of the infusion exactly the inverse of those seen in patients not shocked, and similar in all respects to those following any peripheral irritation in shock. (Chart VI.) The blood pressure begins to fall as soon as local tension is produced, and the fall is greater than any rise seen in normal or unshocked individuals, while it is much more permanent, and may result in the reduction of the shock to a grade where relief, previously probable, becomes impossible. Void of true stimulant action as it is, normal salt solution would seem to take a very subordinate position in the therapeutics of shock. Where its administration seems necessary, it should either be given by rectum, or if subcutaneously, with proper care to avoid its peripheral irritative effects. This can be accomplished by giving it at an indifferent temperature, and very slowly; or better and quite simply, by the addition to it of a few drops of cocaine solution.

Raising the general blood pressure by mere mechanical increase in the volume of the circulating fluid, without any central or peripheral heightening of vasomotor tone, would seem *a priori* to be a very difficult matter, and yet such an action has been assumed to account for the supposititious pressor effect of saline infusions. In view of all the evidence at hand, it is probable that the fluid thus added to the tissues, once it begins to enter the circulation, is usually excreted by the kidneys, skin and lungs as fast as it is absorbed. We can find no reference to any clinical determinations of the actual degrees of blood dilution produced by the ordinary quantities of salt solution as given by infusion, but in so far as blood pressure records may give any hint of this,

it appears unlikely that any great increase in the total volume of blood at any one time occurs even after massive infusions that are absorbed with rapidity. Certainly the rapidity of the increase in the amount of urine, and the diminution in its specific gravity following infusions, would seem to allow but little time for the occurrence of any significant increase in blood volume. Whatever increase in this volume may occur, it is certainly too small to affect in any way, by its mere volume, the tension of the arterial wall, and the "filling up of the vessel," which is said to be felt so often after a successful infusion, must be reckoned among the clinical absurdities. Recently we have had the opportunity to really fill up the vessels in a manner beyond dispute, and to show that however extreme a plethora may be produced, it has no necessary effect on blood pressure unless there is simultaneously true central or peripheral (vasomotor) stimulation. Briefly, the case was one of profound traumatic shock, with absolute suppression of the function of the kidneys, the patient being kept alive by means of continuous intravenous injection of adrenalin solution for fourteen hours after death was pronounced to be imminent. During this time about 6500 cc. of a normal salt solution was introduced into the circulation as a vehicle for the adrenalin, and at the time of death, no urine having been excreted in the interval (the bladder contained about 150 cc.), there was a most marked general anasarca, the fluid having all apparently accumulated in the tissues. It was possible to repeat here Crile's beautiful laboratory experiment, and by regulating the concentration and amount injected of the adrenalin solution, to keep the blood pressure at any desired level. When the adrenalin was discontinued the blood pressure fell within a few minutes to a very low level—which was always about 60 mm. Hg. What interests us here is that it was quite indifferent whether the transfusion was discontinued entirely, or whether plain salt solution was administered either at the usual rate or very rapidly—in both cases the immediate fall in blood pressure was equally rapid and complete. It was impossible, even with excretion through the kidneys in abeyance, so to increase the blood volume, even momentarily, as to produce any appreciable rise in blood pressure, the pulse tension even falling rapidly while relatively large additions were being made to the amount of circulating fluid. If rapid transfusion, under such peculiarly favorable conditions, caused no rise in blood pressure, the slow method of infusion of much smaller quantities of fluid cannot be expected to fill up vessels or raise pulse tension mechanically with any permanency.

(III) Hæmorrhagic hypotension is one of the most emphasized indications for the employment of salt solution, which is here also usually given subcutaneously, its use in favorable cases being always attended by an increasing pulse tension. As noted under the discussion of hypertensive states, loss of blood is among the most rapidly acting of all agents producing a fall in blood pressure, as it is also one of the most transient in its effects. Therapeutic bleeding of moderate amount produces a V in the blood pressure curve, the rising arm being usually the longer, though frequently enough the pressure returns to its high level almost, if not quite, as rapidly as it fell. Hæmorrhage from any accidental or pathological cause has exactly similar results, and the spontaneous tendency of low pressures after hæmorrhage to return towards the normal is well recognized. If a bleeding vessel remains open, loss of blood from it will cease when the blood pressure falls, and will only recur if the recovery in pulse tension is too rapid to allow of the formation of a coagulum firm enough to withstand the increasing hydraulic pressure. If the source of hæmorrhage be absolutely controlled, measurements of the pulse tension will show in every case this descending and then ascending type of curve; and the ascending or returning limb of the V will follow, in the absence of medication, a very regular course.

This physiological curve of pressure, associated with hæmorrhage, makes it difficult to control by means of blood pressure measurements the value of the agents employed to expedite recovery. If the hæmorrhage continues when these agents (other than the purely mechanical procedures for the arrest of bleeding) are employed, the blood pressure will continue its downward course uninterruptedly until arrest is complete. If they are administered after the local control of the hæmorrhage has been accomplished, when the pressure has reached its lowest point or has begun to return, their effects are obscured by the normal post-hæmorrhagic pressor tendency. Given at this period, we have found small doses of morphia to be followed by a rise in blood pressure as great as that following large doses of strychnine in other cases; but certainly the rise in such circumstances could in no way be attributed to any stimulant properties possessed by morphine. The morphine, it is true, produced no break in the amount and rapidity of the rise which was already occurring, and it was easy to tell by an inspection of the chart that the curve was the unmodified physiological one of post-hæmorrhagic return. True stimulants (strychnine, digitalin), on the other hand, are capa-

ble of accelerating this return, and palpably increasing the steepness of the curve. Saline infusions, in this solitary instance, may have a similar effect, and apparently act definitely in increasing the blood pressure. With due avoidance of the *post hoc ergo propter hoc* system of logic, and a consideration of the natural course of events in the absence of any form of treatment, it is fair to conclude that infusion of salt solution may have in hæmorrhagic hypotension a real pressor value. That this pressor effect is the result of stimulation, properly so called, is impossible in view of its constant failure to appear in any other class of cases. It is, then, necessary to admit that in some circumstances, when the blood volume is very markedly reduced, a purely mechanical addition to it of an indifferent fluid may be retained in the circulation long enough to affect appreciably the tonicity of the vessel walls.

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THE VALUE OF TUBERCULIN IN SURGICAL DIAGNOSIS.

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The difficulties in the differential diagnosis between certain surgical affections and tuberculosis often being so great, it is surprising that so few surgeons have availed themselves of the use of tuberculin as a diagnostic aid in doubtful cases. Failure to meet the high expectations of those who early used it as a therapeutic agent has no doubt been partly responsible for this, but a still more important reason for the hesitancy of many to make a trial of its value was the grave charge made by Virchow that its use was likely to start into activity latent foci of disease and even to spread disease to other parts of the body. Notwithstanding this unfavorable opinion, tuberculin has continued to be used by a few and those who have had most experience in its use have, almost without exception, testified as to its harmlessness and its great value as a diagnostic agent. At the recommendation of Dr. E. L. Trudeau, Dr. Halsted has had the tuberculin test applied to most of the surgical cases in the Johns Hopkins Hospital suspected of tuberculosis since 1896. During the first few years it was found to be so free from bad effects and so helpful in the diagnosis of many obscure conditions that its use was continued and it is now employed in all doubtful cases. Since October, 1900, I have made personally over 500 tuberculin injections in over 300 patients in the wards of the Hospital.

This paper is based on a study of the results of injections in 400 cases recorded in our histories. A number of patients under treatment in the medical and gynæcological services, who were injected, are not included, nor are a number of surgical cases so recently injected that nothing very definite can be said of the ultimate results. The most important questions to determine with regard to the use of tuberculin seem to be, first, is its use harmful? and secondly, is the test reliable?

Is the Use of Tuberculin Harmful?

This is by far the most important question for decision. If it be true, as has been maintained by some, that the injection of tuberculin disseminates tuberculosis or arouses latent disease into activity, its use as a diagnostic agent, no matter how reliable it might be, certainly could not be justified. Experience during seven years has led us to believe that in the small doses needed and under proper conditions, the injection of tuberculin for diagnostic purposes is not attended with danger and should not give rise to serious or distressing symptoms. In one-third of the cases the patients were under observation in the hospital twenty-one days or over and one-eighth of the cases were under observation three months or over. A large proportion of the patients return to the hospital for observation and no serious consequence clearly attributable to the use of tuberculin has been seen in the cases personally observed or noted in the histories in this series of cases in which Trudeau's tuberculin was used. If the injection of tuberculin arouses latent tuberculosis into activity or disseminates the disease this should be evident as early as within three weeks after the injection. In nearly all cases the patients are injected within the first few days after admission to the hospital, hence the number of days in the hospital minus two or three days while the temperature is being taken before the injection is made gives a fair estimate of the time these patients are under careful daily observation in the wards after injection.

A limited experience seems to show that all men, whether tuberculous or not, react if the dose be large enough, but patients are usually extremely ill after large injections: the temperature frequently rises to from 103° to 105.5°, headache is intense, and frequently there are nausea and even vomiting as well as backache, chills and general discomfort. Several writers state that they use ten milligrammes as a routine injection, and some have employed as much as twenty or thirty milligrammes, but the use of such large doses for diagnosis is not only entirely unnecessary but defeats our purpose. To avoid disagreeable symptoms we begin with a dose of one to three milligrammes, and if no reaction is obtained the amount is increased at intervals to a maximum dose of nine milligrammes, thus avoiding the very disagreeable symptoms which accompany the use of large doses. None out of one hundred and fifty cases which

later observation seemed to indicate were definitely non-tuberculous reacted to six milligrammes or less, hence any adult patient reacting to six milligrammes or less is considered certainly tuberculous. A reaction to nine milligrammes is strongly presumptive but not positive evidence of tuberculosis.

Virchow and a few of his students seem to be responsible for the quite prevalent belief that the use of tuberculin is dangerous. In a paper which appeared less than a year after the introduction of tuberculin he stated that he had had opportunity to examine pathological specimens from twenty-one patients who had been injected with Koch's lymph. He presented some of the specimens at one of the Berlin societies and stated that he had seen on the serous membranes the eruptions of entirely fresh miliary tubercles under circumstances which made it scarcely probable that they were old. It seemed to him that they must have been formed after the injection and he thought it probable that the softening of caseous areas was responsible for the spread of the disease. It should be borne in mind that these patients had all had repeated injections of tuberculin, given as a therapeutic agent and that the doses commonly used at that time were high. Virchow's observations were limited to comparatively few patients and made within a short time after the use of tuberculin was begun. His statements are much more guarded than many who have criticised the use of tuberculin would lead us to believe. I have not been able to find that Virchow personally ever published any further articles confirming this first adverse criticism. At about this time, now nearly twelve years ago, several of his students also came out with adverse criticisms of tuberculin. Grawitz, from experiments on monkeys, believed that Virchow's impressions were correct. Bardenhauer reported some cases at the German Surgical Congress in 1891 which also supported this view. Mikulicz believed that harmful results might occur unless tuberculin was used with great care, but on the whole seemed to be in favor of its use. Von Leyden from a very limited number of cases, reported two in which long-continued fever followed the use of tuberculin. Apparently there have been very few observations made recently. Knopf doubts the utility and wisdom of using tuberculin for diagnosis, but does not state whether his objections are based on any unfavorable personal experience. Among those who disagreed with Virchow at the time of his publication were von Ziemssen, Guttmann and Ehrlich, Schede, Curschmann and numerous others, many of whom will be mentioned further on in this article.

Is the Test Reliable?

To settle the second important question, the reliability of the test, we must determine: (1) Do non-tuberculous patients react? (2) Do patients with definite tuberculosis ever fail to react?

Reaction of Non-tuberculous Patients.—The only positive means of determining that patients are non-tuberculous is, of course, a careful post-mortem examination. This we did not find opportunity to make except in a very few of our cases. A number of patients that were apparently not tuberculous gave a positive reaction. One patient in particular had a large mass in the abdomen and a diagnosis of probable tuberculous peritonitis with a rolled-up mass of omentum was made on the basis of a positive tuberculin reaction. On the operating table the mass was found to be carcinoma of the stomach and the accuracy of the tuberculin test was very much in question. This patient died a few days later, however, and at the post-mortem examination numerous small, unsuspected tubercles were found scattered throughout both lungs. The tubercles were not large enough so that their presence had been suggested by the physical signs before operation. In this way the tuberculin test may be at times misleading, though really not at fault if it indicates a lesion other than the one to which attention is at the time specially directed. Madison reports a case in which he considered the reaction positive but no evidence of tuberculosis was found at a careful post-mortem examination. I should not consider this reaction positive, however, for the patient did not definitely react to four or seven milligrammes and a reaction to ten milligrammes is not infrequent in healthy persons. Elder reports his experience in the use of Trudeau's tuberculin in a limited number of cases, and does not believe that the reaction is constant in tuberculosis. Three of his cases, thought to be possibly tuberculous, did not react, but afterwards yielded to specific treatment, seeming to indicate that the disease was syphilis. Two cases of tuberculous peritonitis gave no reaction. Kossel, who made a series of injections in children and afterwards had the opportunity to make post-mortem examinations in several cases, found latent tuberculosis of the mesenteric and bronchial glands accounting very frequently for reactions, the cause of which could not be determined before death. Von Jaksch reports six cases supposedly non-tuberculous which reacted to tuberculin. At post-mortem examination one

of these showed an unsuspected tuberculous deposit in the lungs. He does not give further information about the other cases. Barney has reported three supposedly non-tuberculous cases that reacted. Von Leyden states that in his control injections most of the patients did not react but some of them in whom tuberculosis was absolutely not to be suspected did react. Von Bergmann reports reactions in cases of actinomycosis and breaking down sarcoma, though he acknowledges the possible presence of latent tuberculosis in these patients. Kalindero and Babes have reported two cases of leprosy with reactions which lasted for several weeks after the injections of eight milligrammes of tuberculin. Von Eiselsberg also reports a reaction in a case of actinomycosis and Goldschmidt in a case of leprosy. Practically all of these cases were reported during the first year after Koch announced the possible value of tuberculin in the cure of tuberculosis and most of the writers have apparently never continued their observations to determine by further investigations whether they were correct or not. Tuberculin was then the panacea for all ills: it was not only supposed to give reactions in leprosy and actinomycosis, but its use was suggested for the cure of these diseases. The most extravagant reports of wonderful cures by tuberculin are to be found in all the German journals for 1891. If some of these writers were not more careful in their observations of reactions in pathological conditions other than tuberculosis than they were in reporting cures from its use, their statements certainly cannot be considered very reliable. One great source of error was their very short experience in a limited number of cases. Ten or at most fifty cases were considered sufficient to justify a long article about the use of tuberculin. As examples of this: von Jaksch reported a series of only twenty-eight cases, Berthenson thirty-five, Grasset fourteen, Guttmann and Ehrlich thirty, and the majority do not make any definite statements as to the number of their cases, but it is very evident that their observations were limited. For the most part large doses were used, of indefinitely standardized tuberculin, little pains were taken in charting the temperature preliminary to and following injection and the methods were generally careless.

Definite Tuberculosis without Reaction.—In several of our cases tuberculosis was thought to exist but there was no reaction from repeated injections. The tissues sometimes had the macroscopic appearance of tuberculosis at the operation, and in one case three injec-

tions failed to give a reaction, but numerous tubercle bacilli were found in the tissue removed from an abscess over a rib. The demonstration of tubercle bacilli, tubercles seen microscopically, or tuberculosis produced in guinea-pigs by inoculation, are the three positive tests of the existence of tuberculosis which should be present before we say definitely that tuberculosis exists without the patient's giving reaction to the injection of tuberculin. Among the cases of this kind which have been reported may be mentioned that of Port, a German military surgeon, who reports one case in which tubercle bacilli were found in the sputum with no reaction to tuberculin, but he considers a local reaction necessary to give positive proof of disease. Von Leyden mentions a case of advanced tuberculosis demonstrated post-mortem without reaction. A few other cases of this kind have been reported. White estimates the number of these cases at about thirty. Head and Guttsdat from statistics place the margin of error at about five per cent while Madison from a limited number of post-mortems and Fraenkel from 8000 post-mortems in cattle, place the number of errors from this source as high as ten per cent. Practically all of these cases were reported during the first year of the use of tuberculin when everybody was rushing into print either to laud or condemn its use. Many possible sources of error are present in these cases: the use of old, worthless solutions; the uncertainty as to the strength of the stock solutions because of imperfect methods or careless standardizing; leaky syringes; lack of carefully kept two-hour temperature charts; the use of only one dose in doubtful cases in which graduated doses should be used, are among the most important of these.

The Preparation of Tuberculin.—The tuberculin which has been used in our series of injections is that commonly known as Koch's old tuberculin and is furnished to Dr. Halsted by Dr. E. L. Trudeau, of Saranac Lake. Koch's method of preparation was to grow tubercle bacilli on a glycerine bouillon culture until exhausted; the culture medium was then filtered and the filtrate was reduced to one-tenth of its original volume in a water bath at a temperature of 100° C. This heating at high temperature for several hours insures that the crude tuberculin is free from any living germs. The solution is standardized by injecting tuberculous guinea pigs, graduated doses being given. As Koch originally stated, the crude tuberculin seems to keep almost indefinitely without losing its strength. It is not in suitable form for use until diluted, however, and probably no

better way of diluting it has been suggested than Koch's original method of using 0.5 per cent of carbolic acid in distilled water. Koch noted a distinct loss of power in diluted solutions on standing and advised that it should be prepared freshly for use. My experience has been that a diluted solution over two days old is not reliable; for forty-eight hours it can be depended upon, after that length of time it is frequently worthless. In diluting the solution a one-gramme graduated pipette with one-hundredth divisions is used. The amount of tuberculin drawn up in the pipette depends upon the size of the dose it is desired to give. For a three-milligram dose three hundredths of the crude tuberculin is drawn up. This is diluted in a tube graduated in cubic centimeters, with ten cubic centimeters of the diluted carbolic acid solution. This gives ten cubic centimeters of diluted solution, each cubic centimeter of which contains $0.03 \div 10 = 3$ milligrammes. As one cubic centimeter is equal approximately to fifteen minims, it is not difficult to administer the tuberculin with an ordinary hypodermic syringe, fifteen minims of the diluted solution giving the minimum dose. When six or nine milligramme doses are required 9-100 of the crude tuberculin is drawn up in the graduated pipette and diluted with ten cubic centimeters of the dilute carbolic acid solution. This gives a solution, one cubic centimeter or fifteen minims of which contains nine milligrammes of crude tuberculin. Three milligramme doses can be given by taking five minims of this solution: six milligramme doses by using ten minims. In preparing the solutions the greatest care as to aseptic technique should be used, for tuberculin is quite irritating and it would be easy to produce a serious infection or abscess. The bottle in which the solution is kept and the tubes and pipette which are used in preparing it should always be boiled for a few minutes, to make certain that they are sterile. An ordinary hypodermic syringe, asbestos packed, with a glass cylinder, is best for use in the injections. With the glass cylinder one can see if the piston is not tight and any of the solution leaks back of it while the injection is being made. Solid metal syringes which were tried at the hospital for some time were not found reliable. They soon leak and it is impossible to see how much is really injected. On several occasions it was necessary to give a second dose because of a leaky syringe. No doubt a good deal of the difference of opinion that exists as to the proper dose of tuberculin is because of leaky syringes and the differ-

ence in strength of the different crude tuberculins. Otis has emphasized the importance of using only a standard preparation of which the strength is known, and his suggestion is well worth notice. Another possible source of error is in drawing up the tuberculin in the graduated pipette. As the lower part of the pipette is not graduated it is necessary to partially fill the pipette up to the graduation marks with diluting solution. Without care is used some of the fluid is likely to drop from the pipette into the bottle and dilute the stock preparation. Differences in strength are indicated from the fact that Grasset, who uses only two-tenths of a milligramme of tuberculin prepared in Pasteur's laboratory, apparently obtains as positive reactions as we get with two or three milligrammes of Trudeau's tuberculin; while White, Barney and Otis use ten milligrammes of Koch's preparation to give the same reaction.

Tuberculin R, which Koch introduced some time later, and which is made by grinding up tubercle bacilli, was definitely proved to be unsafe. I am not aware that its use was ever advised for diagnostic purposes; it is certainly not used at the present time for any purpose.

The Temperature Chart Preliminary to Injection.—In order to determine that the patient's temperature is not ranging above normal, it should be taken at two-hour intervals for at least eighteen hours before injection. It is probably better that the temperature be carefully watched for even a longer time; Trudeau advises the temperature to be followed for several days before injection. Several who have written on the subject do not consider it necessary that the temperature should be afebrile, but this is no doubt one source of the supposed unreliability of the test. Heron, at the British Tuberculosis Congress, mentioned the neglect of the rule never to administer tuberculin unless the patient's temperature had been normal for at least twenty-four hours, as one of the reasons why the test had fallen into discredit. It is evident that if a patient has a fluctuating temperature from some suppurative process or from æstivo-autumnal malaria, and if the temperature be not carefully observed and elevations recorded previous to an injection, an elevation of temperature after an injection might readily give rise to mistaken diagnosis. The routine which I have adopted is to have a two-hourly temperature chart begun at six in the morning; this is continued until midnight, when the tuberculin may be given. Accidental rises without any assignable cause occurred in a number of cases in which preliminary temperatures were being

taken. If there be no special necessity for haste it is better to watch the temperature for several days, but within eighteen hours any fluctuation of temperature is likely to be detected. After the injection the temperature is not taken until six the next morning, as it has been found by observation of a very large number of cases that patients do not react to a small dose of Trudeau's tuberculin in less than six hours. The usual time is from eight to twelve hours. This gives the patient a fairly good night's rest from twelve at night until six the next morning. Awakening the patient every two hours during the night for the two-hourly temperature, as was formerly done, was found one of the greatest sources of annoyance connected with the use of tuberculin. If the patient has a slight elevation of temperature he should be kept in bed for a few days, when frequently the temperature falls to normal. Anything above 99 degrees Fahrenheit is considered to contraindicate injection. Although tuberculin has occasionally been given to our patients when their temperatures were 100°, the test is not nearly so reliable as if the temperature be strictly normal. The injection of patients with fever was practiced during the early experience at the hospital in the use of tuberculin, but proved unsatisfactory. With Trudeau, Heron and others, I should insist on the necessity of observing the rule, never to give the injection if the temperature be elevated.

Technic of Injection.—As tuberculin is quite irritating and sometimes gives rise to considerable soreness after injection, it is wiser to make the injection deeply into the muscles rather than subcutaneously. In the early use of tuberculin two bad infections arose. In my own series a small area has been prepared on the outer side of the shoulder with ether, alcohol, mercuric chloride solution and sterile gauze. After the skin has been cleaned a small sterile gauze pad is held in place over it by a cross of adhesive straps. The nurse can make the preparation at any convenient time and the pad keeps the area clean until the time of injection. After injection the pad is replaced and allowed to remain for twenty-four hours or so. With this technic there has not been a single inflammation of any serious nature, although in many cases the patients have complained of a good deal of soreness at the site of injection. The injection is usually made directly into the deltoid muscle, but it may equally as well be made into any of the other muscles. Without some antiseptic precautions, it seems almost unavoidable that some trouble will arise in hospitals where there are many sources of infection.

Nearly all who have used tuberculin extensively advise deep injections. Koch first injected subcutaneously, but his early experience led him to advise deep injections; he prefers to inject into the deep muscles of the back midway between the shoulder blades.

The Dose.—Various doses have been used at different times since the routine use of tuberculin was begun. At first one milligramme doses were used. Then the dose was increased to two milligrammes, and if no reaction was obtained doses of four and six milligrammes were given. During 1900 and 1901 these were the doses used as routine, but in 1902, on beginning the use of a new supply sent by Dr. Trudeau, patients did not react to two milligrammes readily, as with the old solution. Hence the minimum dose was changed to three milligrammes, which was found to give about the same effect as two milligrammes of the former solution. Beginning with a minimum dose of three milligrammes it is now increased to six and nine milligrammes if no reaction is obtained with the smaller dose. From our experience it is quite evident that the strength of different samples of tuberculin varies greatly, even when prepared in the same laboratory. The difficulty in standardizing can be readily understood, for guinea pigs which are used to test the strength of the solution undoubtedly differ considerably in their susceptibility. When we find, as is frequently the case, that patients give a marked reaction to the smaller dose it seems unjustifiable to begin with a large dose which would make them very much more uncomfortable. By beginning with the small dose and gradually increasing it, a reaction can be obtained without giving the patient very much discomfort. The general custom of using large doses probably came from Koch's original suggestion that ten milligrammes should be the minimum dose. White, who has made a most thorough study of the use of tuberculin, states that the smaller doses are not reliable and prefers to give but the single dose of ten milligrammes. White's statement seems to me incorrect. The smaller the dose which will give a reaction the more definite the evidence of tuberculosis. It is also certain that some patients, apparently non-tuberculous, react to ten milligrammes, and if the dose be made large enough all healthy persons react. Nearly one-half of our patients reacted to the minimum dose. This being the case, it does not seem fair to subject patients to the discomfort of a larger preliminary dose. Many later writers have come to recognize the importance of small doses. Langenbuch says that high reactions are unnecessary and

harmful and that the dosage should be so regulated that only the necessary elevation of temperature to indicate the disease should occur. By using graduated doses this is perfectly possible. Bandelier also advocates beginning with small doses, which he says should be repeated and increased if necessary. He considers ten milligrammes the maximum dose justifiable. Goetsch says that from a large experience he believes if there is no reaction to five milligrammes the patient is not tuberculous. Moeller, Spengler and numerous others have also advocated the use of small doses. Heron considers the use of large doses one of the causes which has brought the use of tuberculin into discredit. When we consider the difficulties of standardizing and the different strengths of the stock solutions prepared in different laboratories, these differences of opinion with regard to the minimum and maximum doses are readily understood. The influence of location of disease, sex, age, etc., will be considered farther on.

What Constitutes a Definite Reaction?—The most constant result of injection of tuberculin is elevation of temperature with quite a characteristic curve. The temperature usually begins to rise about ten hours after the injection. In a considerable number of cases there is a preliminary fall of temperature, sometimes of two or even three degrees. The temperature then rises quite rapidly for six or eight hours, when the height of the curve is nearly reached; it rises slightly for two to four hours to the maximum, falling a little again for two or four hours, then quite rapidly during the following four to eight hours, but the fall is not as rapid as the rise and frequently there is some slight elevation of temperature for forty-eight hours after the injection. Continued elevations of temperature for several days are quite uncommon, occurring in only three cases of our series. A marked preliminary fall, though not by any means constant, occurring in only one hundred and twelve or a little over twenty-five per cent of our cases, is quite significant when present. In but one of these cases was there any doubt about the reaction. With the elevation of temperature there is, almost without exception, some headache, which is usually slight, but in some cases is very severe; frequently there are severe backache and general discomfort. In the more marked reactions the elevation of temperature is ushered in with a chill; gastro-intestinal disturbances are not uncommon; loss of appetite is usual; nausea and vomiting are occasionally seen; diarrhœa and abdominal pain have been men-

tioned by Weber and others, but not observed in any of our cases. There is usually slight increase in frequency of respiration and in pulse rate which is nearly always proportional to the elevation of temperature.

Local reactions when present give practically positive evidence of tuberculosis, but under ordinary circumstances there is no necessity of using so large a dose as to produce a local reaction. When present there is pain in the affected part, and if it is near the surface swelling, redness, and increase of surface temperature will be noticed. In a few cases there was quite severe pain and a great deal of tenderness about affected tuberculous joints. The local symptoms usually disappear with the fall of the temperature, but in a few cases have persisted for several days. Berthenson believes that there is a relation between the elevation of temperature reached and the duration of the reaction; when the temperature ranges high, the reaction lasts from forty-eight to sixty hours, while when lower the average duration is from twelve to twenty-four hours. This has not been true of the reactions observed in our series of cases, for in fourteen cases ranging above 104° the temperature fell as quickly as in the cases below 102° and the longest time during which elevation of temperature continued was fifty-six hours.

Port considers elevation of temperature alone unreliable evidence of disease, but this is certainly incorrect. Not only are the large doses which give rise to local reactions unnecessary, but misleading and on account of the severe symptoms which nearly always accompany local reactions are undesirable. Kossel does not consider one reaction sufficient evidence of tuberculosis in a child. This is possibly true of certain indefinite reactions, but a second injection does not seem necessary except in doubtful cases. Lauenstein believes that reactions are usually more prompt, both general and local, in case of surgical tuberculosis than in pulmonary tuberculosis. Trudeau holds the opposite opinion. From observation of a limited number of surgical cases having pulmonary tuberculosis I have not been able to observe any special difference in this regard. Esmarch calls attention to the relation between the extent of the disease and the severity of the reaction. This cannot be entirely correct, for in several of our cases the disease was very extensive, yet a reaction occurred only after repeated injections, the temperature curve was not high and local symptoms were not present. The significance of a

preliminary fall in temperature has already been mentioned. Berthenson reports two cases in which there was continued low temperature, considerably below the normal curve of these patients as taken some days before the injection. No such continuous low temperatures have been observed in our series.

Koch's original description of the characteristic reaction to tuberculin as first announced at the Tenth International Medical Congress at Berlin in 1890, describes the typical reaction after excessively large doses, but such severe symptoms should not follow the use of small doses for diagnosis. Koch injected two and a half decigrammes into his own arm and observed that in the course of three or four hours he had a sensation of general weariness, of drawing in the extremities and difficulty in breathing; then there was a violent chill of about one hour's duration with elevation of temperature, which reached 39.6° C.; at the same time he was nauseated and vomited. These symptoms continued for about twelve hours; lassitude and slight pains about the point of injection lasted for several days. With a smaller dose the symptoms were much less severe.

Factors Influencing Reaction.—*Age.*—Children are usually more susceptible to influences causing elevation of temperature than adults, but the dose of tuberculin should not be proportionately smaller; while individual susceptibility varies greatly it seems that slightly larger doses are required in children than in adults. Romberg's experience agrees with ours and also led him to believe that relatively larger doses are necessary to give reactions in children. He does not consider the test as reliable as in adults. Kossel has reported a large series of injections in children and he believes that tuberculosis of the bronchial and mesenteric glands is a frequent cause of reaction in children. These forms of tuberculosis he believes are frequently spontaneously cured. Aside from the proportionately larger dose required and slightly higher temperatures in children no special difference from adults was observed.

Sex seems to have very little influence, reactions appearing as constantly, the temperature curve reaching about the same height and the size of the dose being practically the same in males and females. Ruedi observed that in a few cases which were under his care females reacted less rapidly than males. His statement does not seem to have attracted much attention, as I have not seen reference to it by any writer except White, whose experience agrees with ours, that there is practically no difference in the susceptibility of the two sexes.

General condition has also very little influence upon reaction. Very slender, delicate patients not reacting more readily or markedly than others.

Healed Lesions.—In two cases of tuberculosis of the hip-joint, operated upon for the correction of deformity, definitely healed lesions were found, but both reacted very positively though one of the patients had been well for over eight years. Frazier and Biggs have reported fourteen cases of healed lesions in bone tuberculosis eleven of which reacted to tuberculin.

Location of disease.—Trudeau believes that visceral tuberculosis gives a prompter and higher reaction than bone or joint tuberculosis. Lauenstein holds that reactions are more prompt, both general and local, in surgical tuberculosis. In a limited number of cases of visceral tuberculosis I have not been able to discover any great difference. Tuberculous peritonitis is generally accompanied by such high temperature that I have seldom found occasion to use the test and then it was unsatisfactory, for there was some fever in all cases.

Immunity or Increased Susceptibility Acquired by Repeated Injections.—There can be no question but that patients acquire a certain tolerance to tuberculin, but that this is rapidly acquired by small doses such as should be used for diagnostic purposes is extremely unlikely. To test this I have given repeated doses at intervals to patients who have given definite reactions to the first injection. Without exception a second reaction was obtained. In all of these cases the elevations of temperature did not differ more than one-half degree after any of the doses and there was no evidence from this limited number of cases that small graduated doses for diagnostic purposes gave rise to immunity. White excuses the use of large preliminary doses on the ground that immunity is so rapidly acquired. Von Rück also states that chronic cases soon become accustomed to the poison. Koch states that within three weeks it is possible to raise the dose to 500 times the preliminary dose without severe symptoms. On the other hand, the possibility of increased susceptibility to tuberculin after repeated doses has been suggested by some who have observed the reaction at the Johns Hopkins Hospital, but the experience which I have just mentioned in repeated injections seems to make this also improbable.

A Possible Explanation of the Cause of the Reaction.—If foci of tuberculous disease may be looked upon as large culture

tubes in which the toxines of tubercle bacilli are constantly being manufactured, the explanation of the tuberculin reaction is not difficult. In case the diseased area is small or the tuberculosis in a subacute stage, very little toxine is produced and the system may be equal to the task of taking care of it. If a small additional amount of tuberculin be injected, fever and more or less general discomfort may be produced, for tuberculin is simply a solution of toxines. On the other hand, three or six and usually nine milligrammes of tuberculin when injected in healthy persons are readily taken care of without any fever or discomfort. The amount of tuberculin required to produce a reaction and the intensity of the reaction will depend on the extent and the activity of the tuberculous lesion. Apparently when certain tissues, for example the lymphatic glands, are affected, more toxines are produced by the disease than when bones or joints are affected, and hence a three milligramme dose of tuberculin produces a higher reaction and with greater constancy. When the tuberculous disease is extensive enough so that a great deal of toxine is produced, giving rise to more or less constant fever, the injection of a small additional amount of toxine, for example, three to six milligrammes of tuberculin, cannot be relied upon to produce a reaction. This accounts for the unreliability of the test in patients suffering with fever. I have not noticed that this explanation has been suggested to account for the tuberculin reaction, though it may possibly have been, for it has not seemed worth while to read carefully all of the almost innumerable articles on the subject which have appeared. This explanation seems to me to more satisfactorily explain some of the peculiarities of the reaction than any other. Among these peculiarities may be mentioned the frequent failure of the fever to rise higher in patients with a temperature already elevated; the infrequency of local reactions (provided suitable doses are given) ; the short duration of the reaction. If the reaction be due, as some suggest, to a stirring up of some focus from the influence of tuberculin, it is difficult to see why patients with disease acute enough to cause some fever should not always be still more stirred up and the fever rise higher. It would seem that acute cases would be more readily excited into activity than any others. Then, too, if there is enough local disturbance to cause fever to 103°, why should there not always be local symptoms? Again, if the reaction be caused by irritation of some lesion, why should the elevation of temperature be of such short dura-

tion? When a chronic or latent tuberculous lesion is excited into activity by other causes the disturbance is never so transient. For example, if a tuberculous joint, in which the disease is latent, be injured, lighting up the old trouble, the acute symptoms generally last for weeks or months.

It has also been suggested that tuberculin sets free toxines already manufactured and existing in the body. Just how this is accomplished is not explained.

Most of the early writers do not attempt any explanation of the reaction; indeed, some consider it a very mysterious matter.

Former and Present Opinions Regarding the Value of the Tuberculin Test.—During 1891 nearly one-third of the space in the German medical weeklies was devoted to a discussion of the merits and demerits of tuberculin. In the Deutsche Medizinische Wochenschrift of 1891 alone there are 181 references in the index to articles on this subject. Among the writers at about this time who favored the use of tuberculin are: Curschmann and Ehrlich, who considered it of positive diagnostic value; Naunyn considered the reaction of great diagnostic value, but not absolutely positive; Ziemssen expressed a similar opinion; Riegel considered it a most valuable diagnostic agent, more trustworthy than any other means for early diagnosis, but he believed that some healthy patients react; Esmarch, König, Helferich, Hahn, Braun, Schimmelbusch, Kümmel, and Sonnenburg also spoke in favor of its use. It is not surprising that the pendulum swung in the other direction and that tuberculin fell into general disuse. A few men have persisted in its use, notably those in charge of sanitaria for tuberculosis. The recent statements of some of these men with regard to the value of tuberculin seem worth quoting, for they, of all others, have the large number of cases under observation necessary to determine its value. Trudeau, whose opinion in this country at least is considered the highest authority, says that before condemning the test as at fault when a reaction occurs in apparently healthy individuals, it should be borne in mind that autopsies made on persons dying of other diseases, show some unsuspected tuberculous focus to exist in from 30 to 40 per cent of such cases. He emphasizes the importance of studying the temperature curve for several days preliminary to injection. The injection should not be made in patients who have an elevation of temperature and a moderate dose of not over five milligrammes should be used.

When used in such cases and under suitable precautions he considers it of the greatest diagnostic value.

Spengler of Davos recommends the test most strongly but advises the use of small doses. Petruschky considers it the most certain agent for the early diagnosis of tuberculosis and cites eleven physicians in charge of German sanitaria who favor its use. Bandelier, of the sanitarium in Cottbus, considers it the most trustworthy means of early diagnosis and urges its general use in sanitaria, hospitals and general practice. Goetsch, after an experience of ten years in its use, expresses the belief that if no reaction follows the injection of five milligrammes of tuberculin, no tuberculous lesion is present. Moeller considers its use indispensable in sanitaria and has never seen any bad results from its use. Elder has used Trudeau's tuberculin in a very limited number of surgical cases. He considers it a valuable aid in the diagnosis of tuberculosis of the lymphatic glands, of synovial sacs and bones. He does not believe that the reaction is constant in tuberculous cases, but had no reactions in non-tuberculous cases. No bad effects resulted from its use. Baeri says that it has been used for years in the clinic at Naples for the diagnosis of suspected tuberculosis; in all forms of the disease it has rendered excellent service and no bad results have been observed. At the British Tuberculosis Congress, Heron stated that from observations of some 2000 injections he had never seen evidence in support of Virchow's statement that bacilli are set free by the injection. It has fallen into discredit because of frequent use in unsuitable cases, the administration of too large doses and neglect of the rule never to administer it to patients whose temperature has not been normal for at least twenty-four hours. The prejudice of patients and of the profession is another reason why it has not come into more general use for diagnostic purposes. He thinks there is no other means of reaching a diagnosis so early, and it is without danger to the patient. Koch stated that from an observation of 3000 cases he could say that if properly made the injection is without danger and as a diagnostic test it is almost absolute. McCall Anderson, of Glasgow, considered it a safe and invaluable diagnostic test. Williams disagreed entirely with those who considered tuberculin harmless. France, of Claybury Sanitarium, reported injections of twenty patients free from tuberculosis with no reaction in any of them; in fifty-five suspected cases forty-five reacted; thirty-four of these have since died and in twenty-nine

upon whom a post-mortem examination was made, tuberculosis was found in all. Of eleven still living the diagnosis of tuberculosis is now definite in six. He concludes that the test is of enormous diagnostic value. France's is one of the few series of cases in which after tuberculin injection in man careful post-mortem examinations have been possible.

It is well known that veterinarians have long considered the tuberculin test of greatest possible value. Bang in 515 autopsies in cattle found that the test failed in 9.7 per cent.; Voges reported 7327 autopsies in 1897 with only 2.7 per cent failures to confirm the diagnosis made with the tuberculin test. Bandelier says that even this small percentage is not because of the failure of the test but because of the lack of care at the post-mortem examinations. At the Section on Veterinary Surgery of the British Tuberculosis Congress, DeWar, of the Royal Veterinary College, stated that he considered it the most efficient, reliable and practicable method of diagnosis; Nocard considered the test almost invaluable and Malm almost decisive. It would be an easy matter to refer to hundreds of veterinarians who speak in high terms of the value of tuberculin.

In our series possibly the test has been of the greatest aid in determining the diagnosis in a number of cases in which there was doubt whether we were dealing with hysterical, traumatic or rheumatoid disease of the joints or tuberculosis in an early stage. Several patients were sent out of the hospital without surgical treatment because of the reliance placed on the negative diagnosis furnished by the tuberculin test. Such affections of the hip, knee, spine, etc., are by no means uncommon. It would be a serious mistake to allow early cases of joint tuberculosis to go without appropriate treatment at the only time in the course of the disease when such treatment is likely to prove helpful, and in this class of cases the test seldom fails to give important information. In several cases of tuberculosis of the kidney the test has shown the disease to be present and the diagnosis has been confirmed at operation, in cases in which repeated examinations of the urine have failed to demonstrate tubercle bacilli. No case of tuberculous adenitis has yet failed to give a reaction, and none of the nine cases of Hodgkin's disease or the numerous cases of cysts of the neck, etc., which have sometimes been mistaken for tuberculosis of the lymph glands, have reacted. Only two cases of osteomyelitis out of twenty-five reacted and there seemed to be fair evi-

dence that the disease of the bone was tuberculous in at least one of these. In several of the remaining cases of osteomyelitis there was considerable doubt whether the joint was affected with tuberculosis. None of the cases of actinomycosis reacted to small doses and in one case the dose was raised to forty minims before reaction was produced. No reaction followed injection in several cases of syphilis. In many other cases in which the diagnosis was doubtful and in which early diagnosis was of great importance the test proved helpful, but it seems unnecessary to multiply examples. From personal experience and a study of the writings of others, it seems to me that the evidence at hand is entirely favorable to tuberculin both as regards its harmlessness and its reliability as a diagnostic agent. It remains yet to be proved that tuberculin rouses latent tuberculosis into activity or disseminates the disease, even in the enormous doses in which it was formerly given as a curative agent. The cases supposed to be of this kind are too few in number and the possibility of spontaneous spread of the disease must be remembered. The early writers who questioned the reliability of the test based their conclusions invariably on small numbers of cases, their statements are indefinite and their methods of preparing and administering tuberculin and of charting temperatures were no doubt equally imperfect. Those recent writers who have had most experience in its use are unanimously agreed as to its harmlessness and its high diagnostic value. If properly used in suitable cases I am sure its use will prove most helpful in the diagnosis of tuberculosis from other surgical affections.

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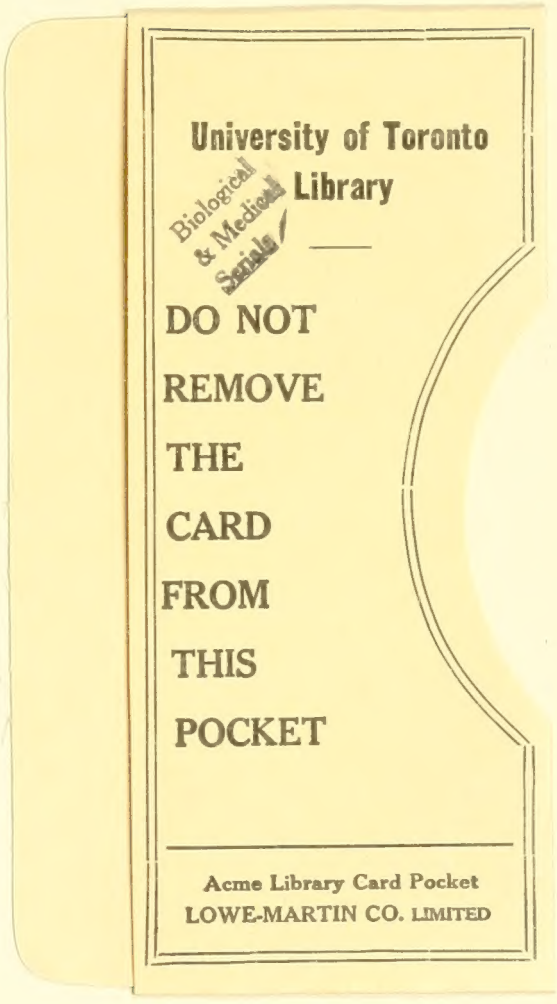
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